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APPOINTMENT OF EDITOR.

We are pleased to announce that Dr. Theodore E. Walsh, of St. Louis, has been appointed Editor of THE LARYNGOSCOPE, effective Feb. 1, 1942.

Dr. Walsh is Head of the Department of Otolaryngology of Washington University School of Medicine, St. Louis, having been appointed to succeed Dr. Lee Wallace Dean upon the latter's retirement from this service.

In 1925 Dr. Walsh received his medical degree from the Medical College of St. Thomas' Hospital, London. His hospital training was received at the Royal Hampshire County Hospital, Winchester, England. He served as Resident and Instructor in Otolaryngology at the University of Chicago School of Medicine and later was appointed Assistant Professor in Otolaryngology there.

Dr. Walsh is a Fellow of the American Otological Society, Inc., and of the American Academy of Ophthalmology and Otolaryngology.

His experience in otolaryngology and his literary attainments qualify him for this position.

NONSECRETING CYSTS OF THE MAXILLARY SINUS MUCOSA.*†

DR. J. R. LINDSAY, Chicago.

The maxillary sinus may be the site of benign cysts of several types. In order to indicate clearly the type of cyst to which this report refers, the following brief classification of the more common types is presented:

1. Benign cysts arising from the jaw or teeth: *a.* Follicular (dentigerous) cyst. *b.* Radicular (root cyst, dental cyst) cyst. *c.* Median anterior maxillary cyst.
2. Benign cysts arising from the sinus mucosa: *a.* Secreting cysts, including gland cyst and mucocele. *b.* Nonsecreting cysts of the sinus mucosa.

The follicular or dentigerous cyst develops from the original enamel epithelium of the tooth. It may be small, or become large enough to fill the antrum completely and encroach on surrounding structures, blocking the nasal passage and nasolacrimal duct. It has a dense fibrous wall, with areas of thin, shell-like bone, and a compact stratified lining epithelium. The crown of the abnormally situated tooth from which it arose protrudes into the cyst.

The radicular cyst arises in inflammatory tissue about the root of an infected tooth. The cyst wall may consist of granulation tissue, in which case it is considered to have developed by central softening, or it may have an epithelial lining derived from epithelial rests of the periodontal membrane.

The median anterior maxillary cyst, arising presumably from epithelial rests at the junction of palate bones and premaxilla, is found in the midline in the region of the anterior incisive canal, extending between the roots of the central incisor teeth. It may bulge upward through the floor of the nose.

*From the Division on Otolaryngology of the University of Chicago.

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Cysts of dental origin are readily diagnosed by the history and an accurate local and X-ray examination.

The maxillary sinus mucosa gives origin to two types of benign cyst, which may be described as the "secreting" and "nonsecreting" types.

"Secreting cysts" include the small gland cysts which are common in the mucosa in the presence of a chronic inflammatory reaction, and the type of cyst known as mucocele. This latter type is frequently found in the ethmoid or frontal sinus, but rarely in the maxillary antrum.

The secreting cysts, whether of the variety of small gland cyst or the larger mucocele, all have an epithelial lining, varying from simple cuboidal to columnar ciliated epithelium. They contain a glary mucoid or a milky opaque type of secretion which does not coagulate. In cases where the secretion has been invaded by organisms and pus cells, the term "pyocele" is commonly applied. The mucocele may develop to huge proportions, invading the orbit and producing extensive deformities of the face. The true mucocele probably develops because of obliteration of the normal outlet of the sinus by inflammatory adhesions, and a history is frequently obtained of a preceding acute inflammatory process with drainage through a fistula, or of a fracture through the sinus.

The nonsecreting cysts of the maxillary sinus mucosa are the most common type. This group has been the subject of special investigation during the past year and will be discussed in this paper.

These nonsecreting cysts form in the connective tissue of the antrum mucosa, and are lined by loose connective tissue, which contains many capillaries and varying numbers of leukocytes and reticuloendothelial cells. They contain a clear amber or straw-colored fluid of low viscosity, which rapidly coagulates after removal. They vary greatly in size, requiring a period of weeks, months, or even longer, to progress to the point where they fill the antrum. They do not cause deformity, like that caused by the untreated follicular cyst, or the mucocele, but rupture spontaneously, with a sudden discharge of clear, watery fluid from one side of the nose. The fluid coagulates in a few minutes, leaving the handkerchief stiff, as if starched. In some instances, rupture of the

cyst, either by puncture or spontaneously, has resulted in its disappearance. In most cases, however, the cyst recurs, either because it is multilocular or because the opening heals over and the cyst refills.

Because these cysts contain transparent fluid, they transmit light freely, hence cannot be detected by transillumination. They appear opaque on X-ray. While small, they appear as a shadow in the lower part of the antrum, with a slightly dome-shaped upper border, in contrast to free fluid in the antrum, which presents a horizontal fluid line, sometimes with a slight meniscus at the edges. As the cyst enlarges, the shadow increases in size until the dome-shaped upper border is lost and the antrum appears diffusely clouded.

Nonsecreting cysts of this type are a relatively common occurrence in the antrum. Some idea of the frequency is indicated by the fact that fluid samples for special determinations were collected from 15 in the space of a year, while still a greater number were either treated or observed during that time without fluid samples having been collected. Because of the fact that local symptoms pointing to the antrum or sinuses in general are often lacking or vague, the presence of a cyst of this type is frequently unsuspected unless sinus X-rays are taken.

McGregor¹ suggested that antrum disease of this type may constitute a focus of infection. Williams and Slocumb,² however, disregarded the antrum cysts because Pattee³ had failed to obtain positive cultures from the cyst fluids.

Our clinical experience has been that the nonsecreting cyst in the antrum may produce a profound train of symptoms. In the case of the very small cyst, there may be little interference with the general health, but as the cyst increases in size a variety of symptoms may develop. In some individuals these appear as general fatigue, low grade temperature elevation, vague headaches, usually in parietal or occipital region, irritability, dizziness on stooping over, postnasal discharge and nasal stuffiness. Head colds are sometimes frequent, but may be absent. Symptoms may develop slowly and progress to the point of total inability to carry on the usual occupation.

In several instances these cysts have been found in individuals with acute exacerbations of infectious arthritis, during

a search for foci. In one individual the rupture of the cyst by antrum puncture was followed in a few hours by chills, fever and acute multiple infectious arthritis, lasting several weeks. Several cases of infectious arthritis who were totally disabled during an acute exacerbation obtained striking relief after removal of the cyst. In some there was a flare-up of joint pains for a few days after operation, followed by complete relief.

Local symptoms, such as stuffiness of the nose, and post-nasal discharge, dizziness on stooping over, are likely to be present, but are usually overshadowed by the general complaints of marked fatigability, headaches, irritability, low fever and, in some cases, pain in joints and acute arthritis. Abstracts of several case reports are given below which illustrate the morbidity caused by this type of disease.

The treatment is relatively simple. When the dome-shaped shadow is evident on X-ray, extending up from the floor of the antrum, a puncture is done. Because the cyst may lie anteriorly, the method of puncture must be such as to permit exploration of the anterior and inferior region of the sinus, as well as posterior. When complete diffuse clouding of the antrum is shown on X-ray, irrigation of the sinus by way of the ostium may in the case of a cyst only produce a few shreds of cloudy mucus. Puncture through the inferior meatus is usually necessary to enter the cyst.

After puncture has been done, the decision as to whether operative removal is necessary will depend upon the severity of the symptoms and upon whether the cyst persists. In some instances, puncture and irrigation results in disappearance of the cyst, and the antrum becomes clear on X-ray. In most cases where the cyst has been large, the relief is only partial and temporary, and is followed by a recurrence. The necessity of the removal of the cyst is then indicated. This the author has sometimes carried out by the radical antrum operation, and sometimes by the simple intranasal antrum window. The latter procedure has been found adequate in all cases where it has been used, and is simpler for the patient. Part of the cyst wall can be readily removed at the window operation, and this is usually sufficient. There is not the tendency for recurrence that is characteristic of an epithelial-lined cyst. The antrum lining has not usually undergone

polypoidal changes in these cases, and does not require removal. If redundant parts of the cyst wall are taken out and the contents completely evacuated, healing without recurrence can be expected. In some instances, simple puncture and evacuation of fluid, and single or repeated irrigation of the antrum has caused the cyst to disappear. If there is a recurrence it can be satisfactorily dealt with through the permanent antrum window.

CASE REPORTS.

Several case records have been abstracted to illustrate the clinical picture. In all of these the cysts could be classed as large, since they appeared to fill at least half the antrum on X-ray and contained several cubic centimetres of fluid.

Case 1: M. H., female, age 65 years. Chief complaints were: 1. Intermittent pain in right knee for two years, recently becoming acute. Confined to bed. Diagnosed as subacute infectious arthritis. 2. During the past two years has suffered from increasing fatigue and has had low grade fever. Had occasional sore throat and two head colds in preceding summer. B. M. R., —19. W. B. C., normal. Local E. N. T. examination was negative. X-ray showed diffuse clouding of left antrum. Antrum window operation done by Dr. T. E. Walsh. There was an exacerbation of pain in the right knee after the puncture of the cyst. Complete relief from symptoms within a few days after operation. Recurrence of mild pains in the knees with weather changes was reported a year later, but no recurrence of the severe preoperative symptoms.

Case 2: E. V., female, age 37 years. Under treatment for duodenal ulcer for five years. Known to have hypertension seven years. Frequent colds began in fall of 1934, lasting until March, 1935, when she was admitted with toxemia of pregnancy, pre-eclamptic. Stillbirth at 28 weeks. Vaginal hysterotomy. For several months previous to operation had complained of facial neuralgia, occipital headaches, fatigue and recurring colds. Some relief after the operation, but she returned in six weeks with complaint of postnasal discharge, tiredness and fatigue. No sign of sinus suppuration on local examination but X-ray showed diffuse clouding of right antrum and a large polyp or cyst on the floor of the left antrum. On May 1 the right antrum was punctured and

about 15 cc. of clear, yellow fluid ran out, followed by a small quantity of mucopus on irrigation. No growth from the cyst fluid on culture. Within six hours patient had a chill, developed a high fever, was then confined to bed for over two weeks with acute infectious arthritis, multiple. W. B. C., 15,000. *Strep. viridans* on throat culture. Following this experience, the patient was loath to permit any further treatment to her sinuses. During the next five months, however, the complaint of tiredness, fatigue and backache persisted. Sinus X-rays five months after the acute illness still showed the rounded shadow in the left antrum, but the right antrum had partially cleared and no soft tissue mass could be seen.

Case 3: A. L., male, age 56 years. Infectious arthritis. Recurrent mild attacks for 10 years. Admitted with acutely swollen and painful right knee and ankle of two weeks' durations. No complaints referable to sinuses. Mild head cold once a year. W. B. C., 9,000. X-ray report: "Grossly thickened membrane in left maxillary sinus with a fluid level." Antrum puncture gave negative results. After consultation it was decided to do a radical antrum operation. Cyst removed; 5 cc. of clear, straw-colored fluid saved for chemical determinations. There was rapid relief of the acute arthritic symptoms after the operation.

Case 4: L. A., male, age 44 years. Gradual onset of temporoparietal headaches, dizziness on stooping over, nervousness, extreme irritability, inability to concentrate, increasing steadily for past 10 months. He would go to his office in the morning, but in an hour or two would have to come home and lie down. B. P., 152/100. B. M. R., —15. Frequent sore throat. Tonsils removed two years previously. Examination of nose and pharynx showed only reddened mucosa in vault of nasopharynx. W. B. C., normal. X-ray showed a shadow in lower half of right antrum with dome-shaped upper margin, and a shadow in lower half of left antrum with upper margin resembling fluid line. Thickened mucosa of lateral wall of right antrum also. A radical antrum operation, left, was done with removal of a cyst filling half the cavity. Antrum window operation on right, without removal of mucosa. Complete recovery from above symptoms after operation. One year later, patient reported having had no return of symptoms.

Case 5: J. S., age 52 years. Patient referred with chief complaints of parietal headaches, bilateral, coming on early

in the day, fatigued very easily, unable to concentrate, irritable, occasional stuffiness in the nose but no colds. Had had recurrent attacks of joint pains, lasting only a few days, for several years. Patient was a hard working, energetic business man but in recent months symptoms had become so severe that he had to give up such activities as golf, and was no longer able to carry on his business. Had been having nose and throat treatments for 10 months with no relief. No X-ray had been made. X-ray examination showed a soft tissue shadow filling all but a small corner of left antrum, and a small dome-shaped shadow in floor of right antrum. Left antrum was punctured through the inferior meatus but no pus or fluid obtained. An antrum window operation was done and a cyst filled with straw-colored fluid removed through the window. The right antrum was not interfered with since the shadow was quite small. Patient had some pains in left wrist and knee during the week following operation. One month later, he had entirely recovered from symptoms. One year later, he reported having been completely well until the past few weeks, when mild headaches had appeared over the vertex and in the back of the head. X-ray showed the left antrum clear but the soft tissue shadow in the base of the right antrum (untreated) was still present and had increased somewhat in size. Culture from the cyst fluid: *Staph. albus* and an anaerobic pleomorphic Gram + bacillus of the bacterioides group.

Case 6: G. H., physician, age 33 years. Recurring sore throat. Headaches — mostly parietal or in the back of the head — tired and fatigued at all times, irritable. Nose stopped up at times. Had been unable to carry on his work satisfactorily in recent months. History of acute sinusitis on left side three and one-half years previously, with chills fever and swelling of left eye. Sudden gush of fluid from left side of nose. No pus was seen in the nose, but the posterior tip of left inferior turbinate was mulberry-like and injected. X-ray showed complete clouding of left antrum. The antrum was punctured and clear, straw-colored fluid evacuated. Symptoms were partly relieved but within a month were as severe as before. Re-examination by X-ray showed the cyst to be still present, half filling the sinus. The radical antrum operation was done and the cyst removed. Fluid saved for chemical determination. Aerobic and anaerobic cultures were

negative. There was purulent discharge postoperatively for several weeks. Patient has experienced complete relief from his symptoms and is a most grateful patient in spite of an unusually stormy postoperative course.

Case 7: H. C., male, age 34 years. Complained of attacks of light headaches, coming on at frequent intervals for past two months, followed by mild dizziness. Easily fatigued in recent weeks. Headaches at irregular intervals, mostly in parietal region. No head colds and no definite complaints referable to the nose. No vomiting with the dizziness. Had recently had a medical examination, which gave negative results. Had been under a heavy mental strain in connection with his business, and complaints were thought to be functional. Psychiatric examination had been advised. Local examination of nose was negative. An X-ray of sinuses was taken and showed a large, rounded soft tissue mass lying in the right maxillary sinus. The antrum was punctured with a straight needle with negative results. When a curved trocar and cannula were used to reach the anterior part of the sinus, several cubic centimetres of clear yellow fluid were collected. Patient was leaving the city and was, therefore, referred elsewhere for further treatment.

HISTOPATHOLOGY.

The gross appearance of the nonsecreting cyst is that of sac with walls thin enough to be translucent. Surrounding sinus mucosa is usually thickened in some areas but seldom shows polypoidal hypertrophy. A small amount of cloudy mucus may be present.

The histopathological picture varies slightly. Figs. 1, 2 and 3 are microphotographs of the cyst wall. They demonstrate that the collection of fluid occurs within the subepithelial connective tissue. No evidence of mesothelium is seen. The connective tissue strands form the lining membrane, with occasionally thin capillaries bordering directly on the fluid contents. In Fig. 1 there are relatively few cells in the subepithelial layers, while Fig. 2 shows large number of eosinophils, plasma cells, lymphocytes and neutrophils. This variation in the cellular content of the cyst wall may be explained on the basis of acute inflammatory reaction. The cyst wall in Fig. 2 is comparable to the section of polyp in Fig. 6, which



Fig. 1.

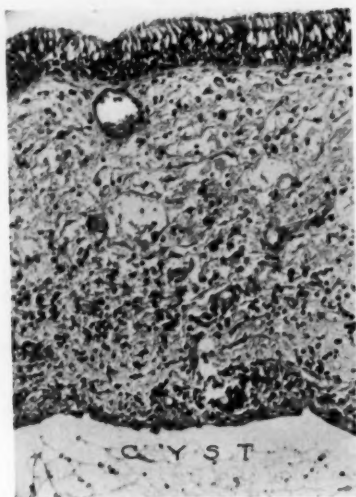


Fig. 2.

Fig. 1 Section of cyst wall showing ciliated columnar epithelium of antrum lining with a thin layer of connective tissue beneath, which forms the wall of the cystic cavity. The cyst has no epithelial or mesothelial lining.

Fig. 2. The connective tissue wall of this cyst shows marked infiltration by eosinophils, lymphocytes, plasma cells and neutrophils. The cyst cavity is not lined by an epithelial or mesothelial layer of cells.

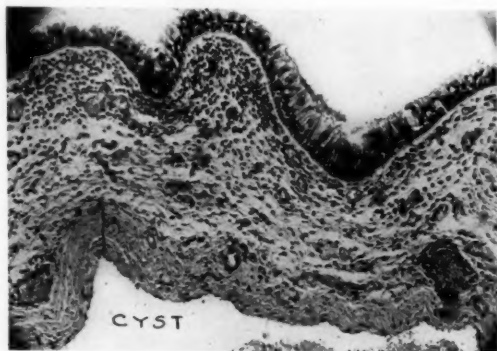


Fig. 3. Cyst wall showing a well defined layer of new connective tissue (A) bordering on the cyst cavity. This probably resulted from organization of fibrinous deposits or hemorrhage at the time of former ruptures.

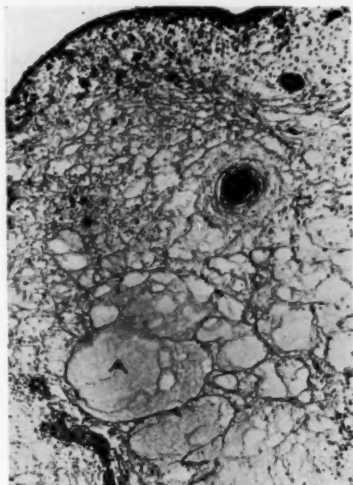


Fig. 4.

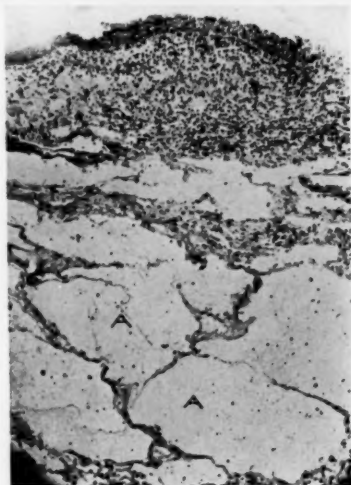


Fig. 5.

Fig. 4. Section from an edematous area in the mucosa adjacent to the cyst shown in Fig. 1 showing microscopic accumulations of fluid, in tissue spaces (A) such as described by McGregor.

Fig. 5. Section from a nasal polyp in a case of allergic rhinitis, showing accumulations of tissue fluid (A) such as in Fig. 4.

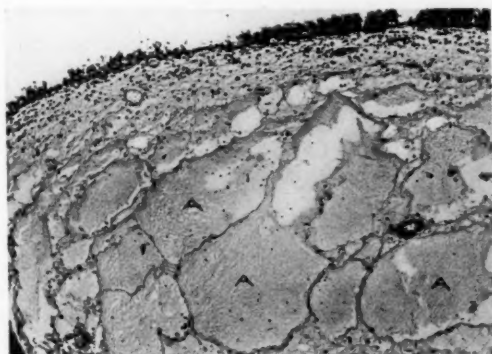


Fig. 6. Section from a solitary polyp removed during acute infection, showing distention of tissue spaces (A) similar to Figs. 4 and 5.

was removed during an acute sinus infection. Neither case could be classed as clinically allergic. Fig. 3 shows a cyst wall in which there has been added a layer of newly organized connective tissue. The cyst had been opened twice and had ruptured several times in the months previous to removal, and had recurred. The new layer of connective tissue probably represents organization of fibrin laid down at the time the cyst had been opened. Fig. 4 shows an edematous area in the adjacent mucosa in the same case as Fig. 1. Figs. 5 and 6 are from nasal polyps showing similar distention of tissue spaces by what is apparently edema fluid. Fig. 5 shows a polyp from a case of allergic rhinitis, and Fig. 6 is from a solitary polyp removed in presence of acute sinus infection. Fluid is seen to distend the tissue spaces in both, although one may be attributed to allergy and the other to infection. In practically all nasal polyps it is possible to find areas which present a similar histological appearance.

McGregor¹ offered the explanation that cysts of the antrum originated from areas such as these by a process of increasing accumulation of fluid in the tissue, breakdown of intervening connective tissue septa and coalescence of small cystic areas thus formed into larger collections of fluid.

This interpretation of the histopathological appearance seems to be possible. Certain questions of fundamental importance still remain to be answered, however.

1. What part, if any, does the allergic reaction in nasal mucosa have in their production? What part does bacterial infection play?
2. Why are these cysts found only in the maxillary sinus?
3. What is the nature of the cyst fluid? How does it compare with tissue fluids in edematous nasal mucosa and polyps? How does it compare with pathological collections of fluid elsewhere in the body?
4. Since there is definite clinical evidence that these cysts may cause systemic disturbances, how is such a reaction to be explained? Is it due to bacterial action, to toxins or to foreign proteins?

The frequent finding of nonsecreting cysts in the antrum and not in other sinuses is explainable on a purely anatomical

basis. Although these cysts have thin walls, they are supported by the floor of the antrum. No matter where they may have originated they gravitate downwards and, hence, have the characteristic appearance of a shadow extending upward from the floor, with a dome-shaped surface.

They are, therefore, protected from stress by the antrum floor and walls until they have reached such a size as to begin to prolapse through the ostium, whereupon sneezing, nose-blowing or other trauma may cause rupture. They do not cause deformity. In sinuses with the ostium in a more dependent part, the action of gravity alone would probably cause prolapse and prevent cyst formation.

These cysts are not clinically characteristic of the allergic reaction. They are more frequent in cases with infection but are sometimes found in the presence of a combined allergic reaction and infection.

The histopathological picture, however, suggests that conditions exist for the formation of these cysts in all nasal polyps, whether allergic or infectious in origin.

BACTERIOLOGY.

The cyst fluids were removed aseptically and cultured for aerobic and anaerobic organisms on ordinary media (blood agar and brain broth). These cultures have been negative in nearly all cases. Cultures from the sinus lumen at the same time as those from the fluids have not as yet been investigated.

In some cases, pneumococci, streptococcus viridans and other organisms have been cultured from the nose of individuals known to have a cyst in the antrum; however, since cysts may be present for a period of months and patients have intercurrent respiratory infections, it is not possible to link the onset of the cyst with a definite infectious process.

The possibility that the cyst originated as a result of acute infection of the mucosa and later persisted and progressed in size, although the infecting organism was eliminated or became attenuated, must be considered.

It appears unlikely that the systemic reaction produced by the cystic fluid is due to bacteria, since symptoms increase in

severity with the increase in the size of cyst and are relieved with elimination of the fluid. Investigations are being carried further in the attempt to determine the reaction of the host to the cyst fluid.

CELLULAR CONTENT OF CYST FLUIDS.

Smears of cyst fluid stained with Wright's stain have shown eosinophils, neutrophils and lymphocytes. The cells were relatively few in most smears, with many degenerated forms. In some, eosinophils and neutrophils were present in about equal numbers. In some, neutrophils predominated, and in others lymphocytes were most frequent.

CHEMICAL ANALYSES OF CYST FLUIDS.

Although nonsecreting antrum cysts are fairly common, chemical analyses of the fluid have never been reported. The fluid for analyses was obtained by direct puncture of the cyst, or from the cyst at the time of operation. The specimens obtained were always clear, with a yellow tinge. The volume of the fluid varied from 2 cc. to 10 cc., depending upon the size of the cyst. After removal of the fluid, clotting consistently occurred. The fibrin was removed by twirling on a glass rod, and the clear filtrate was then used for all analyses.

The number of constituents examined depended upon the amount of fluid available; when the volume of fluid was 5 cc. and over, a complete analyses could be carried out; when the volume of fluid was small, the determinations were rotated to obtain representative data for each constituent.

The following determinations were made on the fluid: pH, total CO_2 , water, protein, chloride, sodium, potassium and calcium. The pH of the serum was determined colorimetrically.⁴ Carbon dioxide was measured with the Van Slyke and Neill manometric gas apparatus.⁵ Chlorides were determined by the wet ashing method of Van Slyke⁶ with the Wilson and Ball modification,⁷ and water by weighing 2 cc. aliquots before and after drying to constant weight in platinum crucibles in a 100° oven. Proteins were determined by the micro-Kjeldahl method of Campbell and Hanna.⁸ The proteins were estimated by multiplying by 6.26 N total nitro-

TABLE I—WATER AND ELECTROLYTE CONCENTRATIONS IN NONSECRETING ANTRUM CYST FLUIDS.

Name	Volume	H ₂ O	Sp. Gr.	Total Protein	Albumin to Globulin	Fibrin	Cl	Na	K	Ca	pH	CO ₂
		gm/L		gm/L		gm/L	mM/L	mM/L	mM/L	mM/L		mM/L
L. A.	7.0	919.3	1.012	65.0			110.4	149.0	4.55		7.64	25.9
H. L.	5.0			51.0			96.3					
D.	2.0	928.8	1.029				98.6		4.94			
L. B.	5.5	911.9	1.024	72.5			81.3	140.0	3.92	2.25		
H. C.				65.1			101.2					
H. P.	2.5			61.4			89.5	141.8				
N. K.	3.0	921.6	1.019	62.7			103.8	142.0				
N. R.	1.0			57.2			100.0					
G. H.	8.5	928.2	1.015	60.0		0.81	109.0	141.4	5.72	2.45		
P. B.	2.5	916.2	1.017	67.4	2.05		101.6		4.43			
A. Z.	3.0	944.6		31.9		0.393	96.7	103.8				
J. H.	2.5	963.4		28.0	1.95		95.7	135.5				
Serum*		937.3		66.6	1.80		100.5	135.5	4.29	2.65	7.35	
Transudates** (Av. values)		967.4		3.09			103.2	139.0	3.34	1.92		
Exudates*** (Pleural)			1.016	41.0			94.6	128.0	5.60	2.22	7.11	15.0

*Hald and Eisenman.¹⁰**Greene, et al.¹¹***Schade, et al.¹²

gen corrected for nonprotein nitrogen. The albumin and globulin were determined by the method of Campbell and Hanna.⁹ Sodium and potassium were estimated by the methods of Butler and Tuthill¹⁰ and Shohl and Bennett,¹¹ respectively. Calcium determinations were made by the method of Kramer and Tisdall¹² as modified by Clark and Collip.¹³

The results of these analyses are presented in Table I.

The points of interest in the data are: 1. The protein content of all cyst fluids analyzed was high. When compared to normal serum the concentration of protein was about the same. When the albumin/globulin ratio in the fluid was compared to the albumin/globulin ratio found in serum, the same average ratio was found. 2. The concentrations of the basic ions, sodium, potassium and calcium were about equal to those found normally in blood serum. 3. The chlorides, with two exceptions, were constantly lower in these cyst fluids than in serum. 4. The specific gravity in five cases of the six determined was above 1.015, and in the other case 1.012.

From this collected experimental data, an attempt was made to determine whether these fluids are transudates or exudates. The differential points to be considered in classifying a fluid as a transudate or an exudate are qualitative rather than quantitative, and since there is no definite line of demarcation, either etiologically or chemically, it is very difficult to make a definite decision.

The differentiation is usually based on the following properties: 1. The specific gravity of transudates is below 1.015, and that of exudates is above; 2. exudates contain much more protein than transudates (3 per cent or above); 3. transudates coagulate slowly, if at all, whereas exudates, because they contain much more fibrinogen, coagulate readily; 4. transudates are sterile but exudates may contain specific organisms.

The findings on these fluids, therefore, suggest that they are exudates, for their specific gravities were mostly high, proteins were high and all fluids coagulated. The pattern of electrolytes, according to chemical analyses, does not resemble that of serum ultra-filtrates — or, in short, a transudate. According to the analyses of transudates by Hastings, *et al.*,¹⁴ and Greene¹⁵ and his associates, the total protein concentra-

tion is less than 3 per cent; the total basic ion concentration is less in transudates than in serum, while the reverse holds for the acid ions, and the water content is on an average of 96.7 per cent. Transudates are in equilibrium with the blood, and the chemical composition is determined by known physicochemical laws. Therefore, when all of the experimental facts are considered, it becomes clear that the fluids from these nonsecreting antrum cysts are not a protein-free ultrafiltrate of serum, but a fluid approaching the properties of a true inflammatory exudate. It can reasonably be assumed, consequently, that the membrane surrounding these fluids has become so altered by noxious influences, or anoxemia, or bacterial toxins, etc., as to permit the free passage of protein so that the concentration of this constituent in the effusion is the same as in the blood.

SUMMARY.

1. Clinical observations have demonstrated beyond doubt that the nonsecreting cysts of the antrum are capable of producing a variety of symptoms. These include fatigue, irritability, headaches, low-grade fever, mild dizziness, pains in joints. Local symptoms of sinus disease may be present or absent. This type of cyst may constitute a focus in infectious arthritis.

2. Nonsecreting cysts are frequent in the maxillary sinus. X-ray examination is necessary to determine their presence.

3. The cyst fluid is usually sterile on ordinary culture methods. This does not rule out the possible rôle of bacteria in the development of the cyst, nor is it necessarily evidence against toxicity. (T.B. pleural effusion and some empyemas have been found sterile. Schade, *et al.*¹⁷)

4. Chemical analyses show that the cyst fluid must be classed as an exudate. This fact suggests that these cysts are not caused by allergic reaction in the mucosa, since in all allergic reactions the resulting edema is a transudate and reversible.

For the production of the cyst fluid, damage to the capillary walls by bacterial toxins, anoxemia or other noxious influences appears necessary. The probable explanation is, therefore, that the cyst originates during an infection and

that the formation is due in the first place to the action of bacterial toxins on capillary permeability.

5. The nature of the reaction, whether due to toxins, foreign protein, or other, which accounts for the clinical symptoms has not yet been determined. Further investigation is being carried out.

The chemical determinations in the above study were made by Dr. Lillian Eichelberger, to whom the author is indebted for valuable aid in the interpretation of the results.

BIBLIOGRAPHY.

1. MCGREGOR, W. W.: *Arch. Otolaryngol.*, 8:505-519, Nov., 1928.
2. WILLIAMS and SLOCUMB: *Arch. Otolaryngol.*, 29:829-834, May, 1939.
3. PATTEE, G. L.: The Relative Value of Various Clinical and Laboratory Methods in the Diagnosis of Chronic Maxillary Sinusitis. Thesis, University of Minnesota, 1933. Quoted by Williams and Slocumb.
4. HASTINGS, A. B., and SENDROY, J., JR.: *Jour. Biol. Chem.*, 61:695, 1924.
5. VAN SLYKE, D. D., and NEILL, J. M.: *Jour. Biol. Chem.*, 61:523, 1924.
6. VAN SLYKE, D. D.: *Jour. Biol. Chem.*, 58:523, 1924.
7. WILSON, D. W., and BALL, E. G.: *Jour. Biol. Chem.*, 79:221, 1928.
8. CAMPBELL, W. R., and HANNA, M. I.: *Jour. Biol. Chem.*, 119:1, 1937.
9. CAMPBELL, W. R., and HANNA, M. I.: *Jour. Biol. Chem.*, 119:15, 1937.
10. BUTLER, A. M., and TUTHILL, E. J.: *Jour. Biol. Chem.*, 93:171, 1931.
11. SHOHL, A. T., and BENNETT, H. B.: *Jour. Biol. Chem.*, 78:643, 1928.
12. KRAMER, B., and TISDALL, E. F.: *Jour. Biol. Chem.*, 48:223, 1921.
13. CLARK, E. P., and COLLIP, J. B.: *Jour. Biol. Chem.*, 63:461, 1925.
14. HASTINGS, A. B.; SALVERSEN, H. A.; SENDROY, J., JR., and VAN SLYKE, D. D.: *Jour. Gen. Physiol.*, 8:701, 1927.
15. GREENE, C. H.; BOLLMAN, J. L.; KEITH, N. M., and WAKEFIELD, E. B.: *Jour. Biol. Chem.*, 91:203, 1931.
16. HALD, P. M., and EISENMAN, A. J.: *Jour. Biol. Chem.*, 118:275, 1937.
17. SCHADE, H.; CLAUSSEN, F.; HABLER, C.; HOFF, F.; MOCHIZUCKI, N., and BIRNER, M.: *Ztschr. Ges. Experim. Med.*, 49:334, 1926.

SPHENOIDITIC HYDROCEPHALUS.*†

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Marked intracranial pressure due to excessive normal cerebrospinal fluid has been found to be at times a complication of otitis. Symonds recognized this syndrome and demarcated it from serous meningitis, and suggested the title of "otitic hydrocephalus," which implied no active inflammatory intracranial process. A similar process can also occur as a complication of sinusitis, and as such "sphenoiditic hydrocephalus" has come into use. The sphenoids thus far have been found to be the causative factor, but since any air space contiguous to the brain may produce the syndrome, "rhinogenic hydrocephalus" is suggested as a more inclusive term.

It is very striking that, although sinus infections are common, a relatively small number develop hydrocephalus; however Williams¹ suggests that from the increasing number of cures of septic meningitis, both through the immediate agency of chemotherapy and other methods, we may expect hydrocephalus to become a problem more frequently. A survey of the literature shows a small incidence of sphenoiditic hydrocephalus. The cases reported are scant, but the fact that I have seen and operated upon two cases within the past two years suggests that this syndrome may have been overlooked. Strauss and Needles² have reported one of these two cases. Carmack reported one case which resembled the course of my cases. Etienne³ recorded a case of intracranial hypertension with papilloedema of sphenoidal origin. Keschner and Savitsky⁴ reported a case in which encephalographic studies were made which showed a definite communicating hydrocephalus.

Symptomatology: The most constant symptoms of rhinogenic hydrocephalus are those due to the hypertensive state: 1. headache and vomiting; 2. blurring of vision and diplopia;

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3. papilloedema. The onset of the disease is usually insidious. The earliest complaints are those of severe headache, which may be occipital, supraorbital or bitemporal. Vomiting, blurring of vision and diplopia soon appear. There may be a bradycardia or the pulse may be within normal limits. Neurological signs of meningeal irritation, such as rigidity of the neck and Kernig's sign, may be evident. More frequently the patient will show stiffness or spasticity of the neck characteristic of sphenoiditis. Marked tenderness of the suboccipital region with zones of hyperplasia along the cervical spine or dermatomes may be present. The characteristic fundus changes, varying from early blurring to marked choked discs, are the most constant signs of this type of intracranial hypertension. Transitory weakness of the abducens may often be noted in the same patient. The pupils may be dilated or contracted and fail to react to light or accommodation.

The disease may last for weeks or months and generally tends to complete recovery. The earlier the recognition and elimination of the diseased focus and relief of pressure of the cerebrospinal fluid by lumbar puncture, the less apt is there to follow optic atrophy or permanent visual deficiency.

Diagnosis: Symptoms of increased intracranial pressure which may be suspicious of an expanding brain lesion, without focalizing signs, accompanied by high pressure of cerebrospinal fluid and absence of cells, in the presence of a sinus infection, point to the diagnosis of rhinogenic or sphenoiditic hydrocephalus. As an aid in differentiating a spreading brain lesion from hydrocephalus, Ayala⁵ formulated his index, which is obtained by readings of the cerebrospinal fluid pressure. It is computed as follows:

$$\frac{\text{Final pressure}}{\text{Initial pressure}} \times \text{amount of fluid extracted (10 cc.)}$$

Strauss, Goodhart,⁶ Savitsky and Keschner have emphasized the value of the Ayala index. They have suggested that where the index is above six, there is likely to be no suppurative intracranial complication. but it rather pointed to a hypertensive intracranial condition. This closely coincides with the theory sponsored by Ayala, who found that in the hypertensive intracranial condition the index varied between seven and 10. The index in one of our cases was 10, while in the

other it varied from two to five. The latter case was not in agreement with those who inferred that in hydrocephalus complicating sphenoiditis or mastoiditis, whenever papilloedema and elevated cerebrospinal pressure are encountered, the index could serve as a presumptive guide in the diagnosis and management of the condition.

In behalf of these authors, it is important to emphasize that the technique employed must be standard and precise. Savitsky has repeated lumbar puncture readings in the presence of physicians who had obtained an Ayala index which did not agree with his hypothesis, and then obtained the correct figures. In performance of the lumbar puncture the position of the patient is important. If the patient keeps his abdomen tense and then suddenly relaxes upon the introduction of the needle, the initial pressure may be too high and then rapidly falls before the next reading, causing a low quotient. The abdomen must be relaxed, the knees must be flexed and the head must be straight. Unless this is carried out, the reading will not be precise. Exercised properly, the Ayala index may help considerably in the differential diagnosis.

Treatment: Whereas "otitic hydrocephalus" may recover spontaneously or respond to repeated drainage of the excessive spinal fluid by lumbar puncture, it appears from our experience that "rhinogenic hydrocephalus" requires surgical removal of the exciting focus for cure. In each of our cases, and the one reported by Carmack,⁷ at the time of operation an empyema of the sphenoid was found with pus under pressure. It is conceivable that extension to the basisphenoid or a bacterial meningitis might supervene if these infections were not drained. Relief of the increased intracranial pressure should also be carried out by lumbar puncture, to be repeated if necessary. If frequent lumbar punctures fail to relieve the hypertension, ventricular puncture may then become necessary for relief of symptoms, as well as for diagnosis.

Comments: Carmack's⁷ case ran a course strikingly similar to ours. His patient was a boy, age 9 years, who complained of blurred vision, diplopia, headache and occasional vomiting after meals, of a month's duration. The vision was 15/20. Examination of the fundi showed bilateral choked

discs of two diopters in the right eye and three diopters in the left. The ophthalmological impression was pseudooptic neuritis or possible brain tumor. The neurological examination showed diminished tendon reflexes and dizziness on being tested for Romberg's sign. There was a slight divergent strabismus. The lumbar puncture revealed clear fluid under 14 mm. pressure of hemoglobin. The nasal examination showed pus in both nostrils and a sinusitis. The patient was observed for three weeks. In this time the papilloedema had increased to three and four diopters. The sinuses were then opened up. Pus was found in the ethmoids, and when the left sphenoid was opened up it was found to be filled with thick gray pus under pressure. The right sphenoid contained swollen mucous membrane. Hemolytic streptococci were found in culture. The eye fields, examined immediately after operation, were found to be contracted. Lumbar puncture showed fluid under 20 mm. pressure of hemoglobin. Three days later the temperature was normal, the headache improved, and from then on there was a continuous but slow improvement. The papilloedema gradually subsided, and two months later the eye grounds were normal and the vision 20/20.

Case Reports: Presentation of the following two cases will demonstrate their clinical similarity and their response to therapy.

Case 1: J. L., a Chinese school boy, age 15 years, entered the Beekman Street Hospital on Nov. 14, 1938, and was discharged Dec. 29, 1938.

Past History: Irrelevant except for occasional severe headaches. He was born in China and lived in this country for two years.

Present Illness: Nine days before admission he began to experience severe bitemporal headache, projectile vomiting, anorexia, fatigue and drowsiness. Four days before admission he complained of diplopia. He had no chills or fever.

Physical Examination: Showed an acutely ill Chinese boy, conscious, co-operative, complaining of severe headache and double vision, which was relieved by closing one eye. The neurological examination by Dr. George Blakeslee revealed slight nuchal spasticity. The pupils were moderately widely dilated and reacted to accommodation but not to light. The fundi were normal. Sinus examination revealed a sphenoiditis. Irrigation of both sphenoids resulted in a return of pus. A lumbar puncture disclosed clear fluid under 180 mm. of H₂O pressure. There were two lymphocytes per cubic millimetre, there was a trace of globulin, the sugar was normally reduced, the smears and cultures were negative and the Wassermann was later reported to be negative.

Course: In the course of the next three days his condition became much worse, the headache was more severe, the nuchal rigidity was marked and partial Kernig's symptoms were present. There was also a

bradycardia, the pulse varying from 46 to 70 per minute. In addition, the pupils now did not react to either light or accommodation. Diplopia was present on looking in all directions. The visual fields and fundi were normal. The diagnosis rested between encephalitis, brain tumor, tuberculous meningitis or meningismus secondary to a sphenoidal infection. It was decided that a sphenoidectomy should be done because of the evidence of pus in these sinuses, with the hope of elimination of that focus as a causative factor in the disease. The operation was performed by one of my staff one week after admission.

There was no subsidence of diplopia, headache or vomiting. The Kernig became definite and the nuchal spasm became more intense. In addition, he complained of pain in his occiput and neck. At the level of his sixth cervical vertebra he was extremely sensitive. An X-ray of this region was negative. He was given sulfanilamide without influencing his symptoms.

Two and a half weeks after admission the fundi began to show haziness of their margins, especially mesially and superiorly. The symptoms persisted and he ran a temperature which reached 101° . A lumbar puncture was done. The spinal fluid was under pressure of 380 mm. of H_2O . The fluid was clear, with six cells per cubic millimetre. Fundus examination on the following day showed complete blurring of both nerve heads and a hemorrhage in the right eye. The opinion was now held that, in the absence of focalizing signs, the evidence of increased intracranial pressure with otherwise normal cerebrospinal fluid pointed to a condition analogous to otitic hydrocephalus of Symonds,⁸ namely, hydrocephalus of sphenoidal origin.

I, therefore, decided to reoperate upon the sphenoid region in order to explore for some unopened recess, osteomyelitis or anomaly which might have been previously overlooked. On Nov. 23, two weeks after the first operation, the sphenoid was revised under local anesthesia. The right sphenoid orifice was found to be just lateral to the septum. On inserting a probe, there was a gush of thick yellow pus. The sphenoid opening was enlarged, the sinus was full of thick creamy pus and extended to the left behind the left sphenoid. The mucous membrane was markedly edematous and congested. Hemolytic streptococci were cultured from the pus.

Daily lumbar punctures were done for a week following the operation. The spinal fluid pressure, which was 520 mm. of H_2O , gradually decreased. The Ayala index varied from two to five.

Ophthalmologic examination following the operation showed a progression of the papilloedema to three diopters in each eye, with hemorrhages in the right. The neck became less rigid, the bilateral Kernig's symptoms persisted but the headache became less severe and the patient generally appeared to be improving. Ten days after the second operation the fundus picture began to show improvement. Three days later only a slight grayish haziness, temporally and above, remained. There was some weakness in convergence and also diplopia.

Three weeks after the operation the discs were normal, the pupils reacted promptly, Kernig's symptoms disappeared, the headaches were gone and the patient looked very much improved. His only complaint was that of diplopia on looking to the left. He was discharged and sent to the country. On his return he was seen in the clinic. He had gained weight and looked very well. Except for a lag of motion of the right eye looking upward, and a residual slight diplopia, which was clearing up, he was normal. Vision in both eyes was 20/20.

A resumé of the important features of this case is as follows:

1. Past history of headache.
2. Present history of intracranial symptoms: headache, vomiting, drowsiness, diplopia of nine days' duration.

3. Nuchal rigidity. Pus on irrigation of the sphenoids. Normal fluid. Lumbar puncture, pressure 180 mm. H₂O, clear fluid, two cells per cubic millimetre.

4. Progression of symptoms including bilateral Kernig's, bradycardia, failure of pupillary reaction.

5. Possibility of encephalitis, brain tumor, tuberculous meningitis, or meningitis symptoms secondary to sphenoiditis.

6. Sphenoidectomy for elimination of known focus, followed by aggravation of symptoms.

7. Two and one-half weeks after admission, beginning blurring, which rapidly advanced to choking of the discs. Lumbar puncture, 380 mm. H₂O, clear fluid, six cells per cubic millimetre. Decision to re-explore sphenoids.

8. Revision of sphenoid two weeks after first operation, finding of unopened right sphenoid with pus under pressure.

9. Daily lumbar puncture for one week, initial pressure 520 mm. H₂O. Low Ayala index. Progression of papilloedema to three diopters.

10. Ten days after operation, beginning rapid recession of papilloedema accompanied by generalized improvement and clearing up of neurological signs.

11. Three weeks after second operation, discs normal. Patient asymptomatic except for diplopia on looking to the left. Discharged from hospital.

12. One month after discharge, fundi normal, vision 20/20, residual resolving slight diplopia.

The case presented a diagnostic problem, especially when improvement failed to take place after the first operation on the sinuses. The typical lumbar puncture findings and the persistence of symptoms of increased intracranial pressure warranted a re-exploration of the sphenoids, which resulted in uncovering a locked-up abscess in the sphenoid. The therapeutic response to proper surgery and lumbar punctures places this in the category of "rhinogenic hydrocephalus."

Case 2: B. G., a housewife, age 29 years, who entered the Mt. Sinai Hospital, June 29, 1937, and was discharged July 18, 1937.

Past History: Irrelevant except for occurrence of frequent colds. She had had maxillary sinusitis in 1934 and 1935. During the latter part of 1935 she had been running a low grade temperature of 100°, which was relieved after her sinuses were treated.

Present Illness: Six weeks before admission, the patient underwent a hysterectomy for a fibromyoma of the uterus in another institution. Cyclopropane, oxygen and ether were utilized for general anesthesia. Three days after the operation she complained of severe headache, stiffness of her neck and dizziness. Examination of the nose showed it to be congested. Both sphenopalatine ganglions were cocaineized without relief. She was then put on acute sinus therapy, consisting of cocaine and adrenalin spray, followed by steam inhalations. Two days later, thin pulsating pus was seen coming from the left sphenoidal fissure. She began to vomit and complained of severe right occipital and supra-orbital pain. Her vision became impaired so that she could see only the outlines of objects. She also developed tingling, numbness and pain in the middle fingers of both hands.

Ophthalmologic examination by Dr. Kaufman Schlivek two weeks before admission to Mt. Sinai Hospital disclosed diplopia upon gazing

in all directions. The visual field appeared grossly normal. There was bilateral papilloedema with hemorrhage and exudate. There was an elevation of three diopters in the right eye and two and one-half diopters in the left.

Neurological examination by Dr. Israel Strauss disclosed glove and stocking sensory disturbances in the upper and lower limbs. There was a zone of hyperalgesia over the third and fourth cervical dermatomes bilaterally. The reflexes were markedly diminished, the knee jerks being almost absent. There appeared to be involvement of central vision. The neurologist did not consider the findings due to an expanding lesion but more probably due to an irritation by a toxic process with involvement of the peripheral nerves, perhaps the spinal roots.

Ophthalmologic examination a week after the preceding examination showed progression. The elevation was one diopter greater in each eye; there were stellate figures in the macular region, and fresh hemorrhages. Three days before admission to Mt. Sinai Hospital a lumbar puncture disclosed fluid under 500 mm. H₂O pressure. After 33.5 cc. of fluid were removed, the pressure was 150 mm. H₂O, yielding an Ayala index of 10.

On admission to the Mt. Sinai Hospital, the patient felt generally better, but vision was unimproved. Irrigation of the sphenoid sinuses resulted in a return of stringy pus from the left sphenoid. Ophthalmologic examination showed an elevation of four diopters in each eye, with several fresh hemorrhages. The cerebrospinal fluid was under a pressure of 380 mm. H₂O. After removal of 10 cc. of fluid, the pressure was 190. The Ayala index was five, there were two lymphocytes per cubic millimetre, and the Pandy reaction was negative.

On July 1 I performed a bilateral sphenothmoidectomy under local anesthesia. The right sphenoid extended to the left side. The mucous membrane was slightly thickened and hyperemic. The left sphenoid was rudimentary, with an empyema of a recess above and mesially, which extended towards the posterior clinoid process. Hemolytic streptococci were cultured from the pus. Two days later the pressure of the spinal fluid was 120 mm. H₂O. The Ayala index was 11.5. A few hours later the elevation of the right disc was found to be two diopters, that of the left eye one and one-half diopters. There were several fresh hemorrhages in both eyes and the change in the macula appeared more marked. Diplopia was not present.

A week later the edema appeared more widespread in both eyes. There were fresh hemorrhages in the right, and the lesion in the left macula had increased. The elevation of the discs was unaltered. Visual acuity of both eyes was 15/30.

Eleven days after the operation the fundus picture was much improved. The elevation of the right disc was one diopter. Both nerve heads were somewhat pale, the margins obliterated, but the peripapillary edema was less, as were the hemorrhages and exudate. The arteries were thin and the veins engorged. The visual fields were likewise improved. Ophthalmologic examination about four months after discharge from the hospital showed a bilateral postneuritic optic atrophy. The vision in the right eye was 15/20 and in the left 15/30. Neurological examination was negative except for the eye findings. The absence of an expanding lesion appeared confirmed and the sinuses were considered responsible for the symptomatology. The patient has made a complete recovery.

A resumé of the important features of the above case are as follows:

1. History of headaches and attacks of sinusitis.
2. Six weeks before admission, hysterectomy under general anesthesia, followed several days later by headaches, stiffness of the neck, dizziness, vomiting, impaired vision, tingling and numbness of hands.

3. Two weeks before admission, diplopia, bilateral papilloedema, three diopters in right eye and two and one-half diopters in left eye.

4. Neurological examination showed hyperalgesia over third and fourth cervical dermatones, diminished reflexes and involvement of central vision.

5. One week before admission, papilloedema was one diopter greater in each eye.

6. Three days before admission, lumbar puncture, fluid under pressure 500 mm. H₂O; high Ayala index.

7. Sphenoids irrigated on admission to hospital contained pus. Fundus examination, elevation of four diopters in each eye, macular edema.

8. Bilateral sphenothmoidectomy, empyema of recess of sphenoid, followed in two days by decrease in papilloedema to two diopters in right eye and one and one-half diopters in left eye.

9. One week later, edema more widespread in both eyes, with fresh hemorrhages.

10. Eleven days after operation, fundus picture improved, elevation right disc one diopter. Marked improvement symptomatically.

11. Examination later disclosed postneuritic optic atrophy. Vision in the right eye 15/20 and in the left 15/30. Neurologically negative otherwise.

The case simulated a brain tumor, the hypertensive fluid, absence of cells, definite sphenoiditis, removal of the focus by operation, and recovery places it in the class of rhinogenic hydrocephalus.

DISCUSSION.

The normal mechanism of the formation of spinal fluid by the choroid plexus, and the absorption of spinal fluid by the arachnoid villi and perineural lymphatics can be altered by a toxic focus. As a result, hypersecretion of excessive spinal fluid may occur, or absorption may become defective, producing increased intracranial pressure, causing hydrocephalus. It is clinically apparent that infection in the sinuses, particularly the sphenoid, may act as the disturbing factor. It is difficult to explain why the vast majority of cases develop a reactive cellular meningitis and only a few hydrocephalus. The possibility of angioneurotic mechanism as a factor in otitic hydrocephalus was held by Quinke.⁹ Passot,¹⁰ who has many adherents, believed that a congenital or chronic lesion which occluded the foramina of Magendie or Luschka or of the aqueduct of Sylvius were the contributing agents. Ersner and Myers¹¹ suggested that venous stasis of intracranial circulation may prevent absorption of the cerebrospinal fluid, and believed this to be clinically manifested when otitic hydrocephalus followed obliteration of the

lateral sinus. Interference of the intracranial circulation does not appear to be a factor in rhinogenic hydrocephalus.

Summary: Two cases of "sphenoiditic hydrocephalus" are presented. The syndrome is similar to that of "otitic hydrocephalus," so named by Symonds. "Rhinogenic hydrocephalus" is suggested as the more inclusive term because any of the nasal accessory sinuses closely situated to the brain may produce this clinical entity. The few cases that have been reported in literature have been of sphenoidal origin. Our cases and most of those reported have had an empyema of the sphenoid. Symptoms suggesting an expanding brain lesion, with hypertensive cerebrospinal fluid, absent pleocytosis, a high Ayala index, and absent focalizing signs in the presence of suppuration in the sinuses, point to the diagnosis of "rhinogenic hydrocephalus." Elimination of the toxic focus by proper nasal surgery, the relief of intracranial pressure by lumbar puncture, will produce favorable results. The paucity of case reports makes one believe that this symptomatology has not been universally recognized.

BIBLIOGRAPHY.

1. WILLIAMS, HENRY: The Underlying Factors Concerned in Otitic Hydrocephalus. *Ann. Otol., Rhinol. and Laryngol.*, 47, Sept., 1938.
2. STRAUSS and NEEDLES: Optic Nerve Complications of Accessory Nasal Sinus Disease. *Ann. Otol., Rhinol. and Laryngol.*, 47:989-1019, Dec., 1938.
3. ETIENNE, ROBERT: Symptomes Oculaire et Orbitaires dans les Sinusitis Sphenoidales." *These de Paris*, p. 39, Case 13, 1921.
4. KESCHNER and SAVITSKY: Otitic and Sphenoiditic Hydrocephalus; Value of the Ayala Index. *Jour. Mt. Sinai Hosp.*, 4, Nov.-Dec., 1938.
5. AYALA, G.: Über den Diagnostischen Wert des Liquordruckes und eissen Apparat Zu Seiner Messung. *Ztschr. f. d. des Neur. u. Psych.*, 8:42-98.
6. GOODHART, S. P., and SAVITSKY, N.: Otitic Hydrocephalus. *Arch. Neurol. e Psychiat.*, 32:1345-1934.
7. CARMACK, J. W.: The Relation of Increased Intracranial Pressure to Disease in the Pneumatic Spaces. *Ann. Otol., Rhinol. and Laryngol.*, 42:264-331, June, 1933.
8. SYMONDS, C. P.: Otitic Hydrocephalus. *Brain*, 54:55-71, April, 1931.
9. QUINCKE, H.: Über Meningitis Serosa Sann. Klin. Vogt. Volkmann, N. F., N.167 (Inner Med. 23), 1893.
10. PASSOT, RAYMOND: Meningitis et Meninges Aseptique d'Origine Otique. *These de Paris*, No. 247, Paris G.
11. ERSNER and MYERS: Otitic Hydrocephalus, with a Suggestion as to Etiology. *Ann. Otol., Rhinol. and Laryngol.*, 45:555-567, June, 1936.

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SPONTANEOUS RUPTURE OF THE LATERAL SINUS IN OTITIS MEDIA.

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Spontaneous rupture of the cranial sinuses following otitis media, although of very rare occurrence, has been known since 1833,¹ when the first case was reported. From that time until 1869 nine cases were observed. Eulenstein,² Lebram³ and Bruhl⁴ collected 23 cases up to 1905, occurring in the following order: Lateral sinus, 17; superior petrosal, one; inferior petrosal, one; carotid sinus, one; bulbar, two; and one case of multiple sinuses. Thus, we can see that all sinuses connected with the temporal bone can spontaneously rupture, but the lateral sinus is by far the one to do so most frequently. It may occur before or after operation on the mastoid and in both chronic and acute infections of the middle ear. Eulenstein² claims that it occurs more frequently in the cases of chronic infection, and the right side is more often involved than the left.

Koerner⁵ explains that the right sigmoid fossa in 77 per cent of the human race is more highly developed than the left. Therefore, the right sinus, being more deeply imbedded in the bones (1.05 mm.) has greater surface contact with the bone and is covered by more perisinus cells. Because of this anatomical structure, the right sinus is more subject to infections than the left. Among 15 cases reported by Eulenstein,² 10 ruptures were of the right sinus.

Postoperative hemorrhage may occur at any time. Cases of bleeding have been reported immediately following operation, four weeks after operation, and even as late as one year after operative procedure; however, the most frequent time for rupture is about one week after operation. These hemorrhages may recur. In cases of acute mastoiditis, elevation of temperature usually accompanies the hemorrhage. These cases often present the clinical picture of otogenic septic pyemia. Metastases, even metastatic retinal hemorrhages have been reported.

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Among the spontaneous ruptures of the acute otitis media type, the scarlet fever and the streptococcus infections play the greatest rôle. The chronic type is usually due to tuberculosis or cholesteatoma.

The sinuses are so constructed that their walls are relatively weak. Being intradurally situated, their lumen is enclosed by the internal and external sheet of the dura, reducing its resistance by one-half. Because of this, it would be expected that spontaneous rupture should occur more frequently; however, a quickly obstructing thrombosis is usually formed in the diseased blood vessel, which blocks the blood stream and prevents such a rupture.

Where a thrombus is not formed, Smith⁶ and Eulenstein,² in accordance with the teaching of MacCallum,⁷ claim that the infection generally advances from the periphery inward through the wall of the sinus. Necrosis of the sinus wall follows when the intima and its endothelium are not traumatized by bacterial action, and it becomes so weak that it actually protrudes and ruptures from the pressure of the blood stream. Such cases were observed by Smith.⁶ In some cases the thrombus in the sinus is produced through a local tributary vessel which becomes infected from the surrounding bone. This, likewise, is not a blood-borne infection but an extension from the exterior incorporating the small vessel. This little thrombus may grow into the lateral sinus which traumatizes from within the vessel wall and a large thrombus may form. This thrombus may soften and open through the infected wall of the sinus. Two such cases were observed by Smith⁶ and two others were reported by Friedenwald.⁸

The bleeding from the ruptured sinus may be external, in which case it goes through the cavities of the temporal bone into the ear canal and the nasopharynx. In operated cases, bleeding occurs through the mastoid cavity as well. If the bleeding is internal, then the hemorrhage travels through the posterior or middle cranial fossa. Of course, external and internal bleeding may be combined.

Prognosis: The prognosis is rather doubtful. Spontaneous ruptures occurring before operation have always proved fatal, while after operation the prognosis is not so bad, although until recently statistics showed a mortality rate of over 50

per cent. This was before the advent of chemotherapy. When the hemorrhage can be seen and controlled and infection likewise combated by the use of the new drugs, the prognosis should be more favorable. Flynn⁹ reported a case in 1933 and cited two other cases observed by Williams and Hill, all of which recovered. Smith,⁶ also in 1933, reported four cases with favorable results in all but one, which was bilateral and followed scarlet fever. This one case occurred before the use of chemotherapy.

As regards treatment, it is obvious that the sinus must be packed to stop the bleeding and the proper chemotherapy should be instituted immediately. Some have ligated the inter-jugular vein, compressed the sinus centrally. But this was all before chemotherapy of sulfonamide was known.

One case which we observed recently was a child, age 4 years, who came under our care in March, 1940, with a bilateral otitis media following an acute tonsillitis. The ears were running a rather mild course, the membrane had to be opened several times, discharge was scant, patient had pain and fever up to 102° F. Sulfanilamide was given but the disease did not subside, in spite of the fact that cultures from the ear showed streptococcus hemolyticus. Bilateral mastoidectomy was performed March 29, 1940. The lateral sinuses were exposed on both sides at the upper posterior angle of the wound at the knee. Both mastoids were completely necrosed. The wounds were left widely open and dressed as usual. Patient had a good deal of reaction, with temperature of 105° F. on the following day. This temperature subsided gradually to normal the next morning; however, the temperature rose again the same afternoon to 104° without a chill. Third day, temperature was 102.6° F. and then it remained at about 102° F. Wounds were dressed daily. Moderate amount of discharge was present. On the seventh day, when dressing was removed from the left side, a tremendous amount of venous bleeding occurred from a rupture of the sinus at its bend, where it had been exposed. Bleeding was controlled by packing. The right side was likewise dressed, with the same results. Bleeding was profuse but was also controlled by packing. Heavy doses of sulfanilamide were instituted and kept up for one week. The culture from the mastoid at the time of operation showed hemolytic streptococci, beta. The

patient's temperature came down gradually. The right side ceased bleeding after four days, and the left side after six days. He then made an uneventful recovery, although the wounds were slow in closing. The patient was discharged from the hospital on April 13, 14 days after operation, none the worse for his experience.

Another case of spontaneous rupture of the sigmoid sinus came under our observation in 1932. Patient was a child, age 6 years, with a bilateral acute otitis media, streptococcus infection. Two weeks following onset, despite early paracentesis and profuse drainage, mastoids remained tender and temperature elevated to 104° F. Bilateral mastoidectomy was performed, both sigmoid sinuses were uncovered. The walls appeared to be normal. The bony cells were filled with pus but no necrosis was seen. Patient seemed to make an uneventful recovery and became completely afebrile. On the thirteenth day, however, bleeding occurred from both wounds, more profuse on the left side. The temperature was suddenly elevated to 104° F. and the child appeared pale, prostrated, had coated tongue. In dressing, the right sinus wall could be seen and the point of rupture was evident. This was small. The other part of the sinus wall showed good healthy granulations. Bleeding seemed moderate in amount and this was stopped by filling the wound with iodoform gauze. The left sinus when uncovered showed itself to have ruptured a greater area and bleeding, consequently, was profuse. This, too, was stopped by packing. Bleeding stopped for two days but temperature remained high. The left sinus bled again; the right sinus was closed. In order to arrest the bleeding from the left sinus, a tampon was placed between the horizontal part of the lateral sinus and the bone, and jugular ligation was performed. Slight amount of oozing continued and iodoform packs were inserted. When bleeding stopped completely, the sinus was explored and a large hole was found in the sinus wall. No thrombus was present and the endothelium looked normal. Patient recovered.

A case of chronic bleeding of the right sigmoid sinus came under my observation in 1922. Patient had a mastoidectomy performed in 1921, the wound of which was not completely healed. When she came to me, examination revealed a retro-auricular opening with tampon in it. When the tampon was

removed, blood gushed out. This bleeding was easily controlled by placing another tampon on the sinus. Patient informed me that such bleedings sometimes occurred when the wound was dressed. The operating surgeon reported to me that the wall of the sinus during operation was uncovered but was not infected and not injured. In my opinion, this sinus had an abnormally thin wall which opened because of slight manipulations.

Conclusions: Spontaneous rupture of the cranial sinuses connected with the mastoid bone may occur, although very rarely. Where it occurs before operation, nothing can be done and patient usually dies. If it occurs after operation and bleeding can quickly be controlled, the patient will usually get well, particularly since the advent of chemotherapy. Three such cases have been reported by us; two acute cases, bilateral; one treated with the proper medicine, and no complications occurred, and the other treated with ligation and no medicine. This case ran a very stormy course but got well without further complications. The third case may not belong here, but it is interesting to know that recurrent hemorrhage can occur in a weak sinus wall without any complication.

REFERENCES.

1. SYME: *Jour. d' Edinbourgh*, 1833; cit. by Gidon These de Paris, 1877.
2. EULENSTEIN: Über Blutungen infolge Arrosion der Hirnblutleiter bei Eiterungen im Schlofenhein. *Ztschr. f. Ohrenheilk.*, 43:29 ff., 1903.
3. LEHRAM, PAUL: Über Spontanblutungen infolge Arrosion des Sinus transversus bei Schavenh otitis. *Ztschr. f. Ohrenheilk.*, 49:77 ff., 1905.
4. BRUHL: Bemerkungen zur radical operation. *Monatsschr. f. Ohrenheilk.*, March, 1905.
5. KOERNER: Die Otitischen Erkrankungen des Hirns.
6. SMITH, ANGELO J.: Spontaneous Perforation of the Sigmoid Sinus Following the Mastoid Operation. *Arch. Otolaryngol.*, pp. 17-43, 1933.
7. MACCALLUM, W. G.: A Textbook of Pathology, p. 13, 2nd Edit., Philadelphia, 1920.
8. FRIEDENWALD HARRY: On the Efforts of Nature to Cure Septic Thrombosis of the Lateral Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 22:1090, 1913.
9. FLYNN, J. A.: Spontaneous Rupture of the Sigmoid Portion of the Lateral Sinus. *THE LARYNGOSCOPE*, 43:65, Jan., 1933.

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THROMBOPHLEBITIS OF THE CAVERNOUS SINUS OF OTITIC ORIGIN—WITH REPORT OF CASE.*†

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Thrombophlebitis of the cavernous sinus of otitic origin is fortunately not a very frequent complication. This is particularly true when it is present as a sole complication. It occurs, however, more frequently when it is associated with thrombosis of the sigmoid sinus or with meningitis. Jansen,¹ in 1893, and Hessler,² in 1896, reviewed large series of cases of otogenic sepsis; both found the cavernous sinus affected in about 5 per cent. On the other hand, a number of investigators both here and abroad, who included in their series only autopsied cases, observed the frequency of otitic cavernous sinus thrombosis to be considerably higher. Thus, Uta,³ in 119 cases of sinus thrombosis, found the cavernous sinus involved in 19, or about 16 per cent. Dwight and Germain⁴ assembled from the literature 149 cases; 43, or 29 per cent, were of otitic origin. Eagleton,⁵ in 1926, reported 15 cases, six of which, or 40 per cent, originated from middle ear sup-puration. Brunner,⁶ in 22 autopsied cases of otogenic sepsis, found a suppurative thrombophlebitis of the cavernous sinus in 12, or about 55 per cent, 11 of which were of otitic origin. Brunner attributes this wide disparity in figures to the fact that thrombosis of the cavernous sinus of otitic origin often remains undiagnosed clinically, little or no physical signs and symptoms being present throughout the entire illness. Even on postmortem examination the condition is not infrequently overlooked, since outwardly the vessel wall may appear normal, and only on incision is a suppurative process disclosed.

The most common routes of extension of infection from the middle ear or mastoid to the cavernous sinus is by way of: 1. the inferior and superior petrosals (usually secondary to thrombosis of the sigmoid sinus); 2. the meninges secondary to meningitis; 3. the carotid plexus of veins. This

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last route of extension has perhaps not received the attention it deserves, in spite of the fact that the intimate relationship of the carotid plexus to the cavernous sinus has been known since 1858, when it was first described by Retorzik (quoted by Koerner⁷).

The carotid plexus of veins in turn may be involved: 1. directly from a lesion in the jugular vein or bulb; 2. secondary to a suppurative process in the tympanum and Eustachian tube; and 3. secondary to a suppurative lesion in the apex of the petrous pyramid, the infection from the last two sources extending through dehiscences, communicating capillaries in the carotid canal, or by means of bone necrosis of the latter. Koerner⁷ was perhaps the first to draw our attention to the fact that an infection in the middle ear may reach the carotid plexus and from there extend to the meninges or intracranial contents. Isolated cases depicting such extension, with or without microscopic sections, have subsequently been reported.^{8, 9, 10}

The following case report concerns itself with thrombophlebitis of the cavernous sinus secondary to suppuration in the apex of the petrous pyramid, extension of the infection having taken place by way of the carotid plexus of veins.

CASE REPORT.

B. M., No. 362283, female, age 52 years, was admitted to the otologic service with the following history: She was well until six weeks ago, when she began to complain of severe pain in the left ear. The pain continued for 10 days, at the end of which time a myringotomy was performed, which afforded the patient relief for the next four days. The pain in the ear, however, returned but was then accompanied by pain in the mastoid region. A mastoidectomy was performed in another institution three weeks ago and the patient was discharged at the end of one week, apparently well. Nine days ago she experienced frontal and vertical headache; three days ago, edema, redness and proptosis of the left eye appeared, followed on the next day by similar signs in the right eye. For the past two days there was a rise in temperature to 104° F., associated with a shaking chill and vomiting.

Examination: The patient appeared acutely ill. There was bilateral proptosis of the eyes. The conjunctiva in each eye was injected and chemotic, and the sclera was reddened. There was complete immobility of the eyeballs. The right pupil reacted well to light, the left sluggishly. The fundi showed virtually no involvement except for slight dilatation of the veins. The nerve heads were not blurred. There was a profuse discharge both from the left middle ear and postauricular wound. The drum was full, the vessels injected. A small amount of mucopurulent secretion was present in the right and left middle meatuses of the nose. Both sphenoids on probing disclosed no pus. Neurological examination revealed equal and active superficial and deep reflexes. There were no

pathological reflexes. The remainder of the examination was essentially negative. Blood pressure was 138/82; blood count: 12,200 white cells, 84 per cent of which were polymorphonuclear leukocytes, 12 per cent lymphocytes, 2 per cent monocytes.

Clinical Course: A culture made from the middle ear discharge revealed bacillus Friedlander. The same organism was obtained from the blood, urine and, subsequently, from the spinal fluid. Meningeal signs: rigidity of the neck and bilateral Kernig sign set in two days after admission. Examination of the spinal fluid revealed 10,200 cells, of which 86 per cent were polymorphonuclear leukocytes. Though there was progression of the eye signs for two or three days, it was followed by a period of regression of these signs for the next four days. The meningeal signs gradually increased in severity, the patient went into a state of stupor and died one week after admission.

Necropsy findings revealed profuse purulent meningitis. The meninges were thickened, a greenish purulent exudate covered the superior and inferior surface of the brain. All of the cranial nerves were surrounded to some extent by the same exudate. The wall of the cavernous sinus on each side was markedly thickened and yellowish. A thrombus, however, was not present within the lumen. The other venous sinuses of the skull opened for examination disclosed no evidence of disease. The ethmoids and sphenoids were found to contain a small amount of thin purulent exudate.

Histologic Examination of the Temporal Bone: There is evidence of a previous mastoidectomy. The mastoid cavity is replaced by suppurative granulation tissue undergoing fibrosis. The tympanic cavity and Eustachian tube are occupied by organized purulent exudate. The mucosa lining these cavities is considerably thickened and polypoid in nature. The ossicles are embedded in granulations and fibrous tissue. There is a large perforation in the tympanic membrane. The inflammatory process has extended into the petrous pyramid, which is relatively pneumatic in character. Within these pneumatic spaces are seen free purulent exudate, granulations, fibrous tissue and new bone formation. The mucosal lining is unduly thickened. The apex of the petrosa, which is partially pneumatic and partially diploic, is involved even to a greater degree. Here there are abscess formation and considerable necrosis with sequestration of the intercellular septa and of the walls of the marrow spaces. The inflammatory process in the petrous pyramid has eroded the carotid canal (see Fig. 1) and has extended to the carotid plexus of veins (see Fig. 2). The latter is markedly engorged, showing evidence of hemorrhagic, perivascular infiltration and thrombophlebitis. The infectious process in the carotid plexus of veins can be traced medially to the cavernous sinus. Newly formed bone is seen lining the carotid canal. It has encroached upon the wall of the carotid artery, having replaced in part the adventitial and medial layers (see Fig. 3). In addition, multiple calcareous deposits with degenerative changes are present within the vessel wall (see Fig. 2). There is no thrombus within the artery itself. The sigmoid sinus, jugular bulb and the superior and inferior petrosal sinuses are likewise free of thrombosis. There is evidence of a recent serous labyrinthitis; multiple deep extradural abscesses where the cortex of the petrosa was eroded; and a meningitis with purulent exudate in the internal auditory meatus, Fallopiian canal and about the Gasserian ganglion.

COMMENT.

This case presents interesting features from the clinical as well as from the pathologic standpoint. After a three weeks' history of acute otitis media, the patient had a mas-

toideotomy performed in another institution. She apparently made an uneventful recovery and was well until three days before admission, when the typical signs and symptoms of cavernous sinus thrombosis appeared. While this diagnosis was readily established clinically, the origin and route of

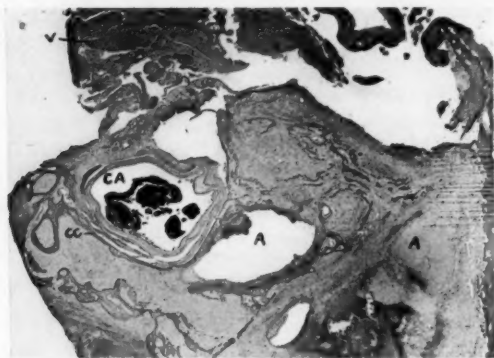


Fig. 1. Section through the apex of the petrous pyramid showing large abscess cavity within and necrosis of carotid canal by direct extension. (A) represents abscess, (CA) carotid artery, (CC) carotid canal, (V) fifth nerve.



Fig. 2. Photomicrograph. Section through the vertical portion of the carotid canal showing thrombophlebitic process in the carotid plexus of veins. Degenerative changes with calcarious deposits within the wall of the carotid artery can be seen at (D). The jugular bulb below is relatively free of disease. (The upper part of the wall of the carotid artery has been partially stripped away from its attachment to the carotid canal-artefact.) (C) represents cochlea (extreme medial tip), (CA) carotid artery, (JB) jugular bulb, (T) thrombus.

extension remained questionable. The accessory sinuses having been more or less definitely ruled out, the condition was attributed to the ear in spite of its relatively infrequent occurrence with infections of the latter. It was further suspected that the infection spread to the cavernous sinus by way of the petrosals, the latter being involved either from the sigmoid sinus or directly from the petrous pyramid. Histologic examination of the temporal bone subsequently proved



Fig. 3. Section through vertical portion of the carotid canal showing new bone formation which encroaches on the adventitial and medial layers of the wall of the carotid artery. Note the inflammatory process in petrous pyramid adjacent to this at (A). (CA) represents carotid artery, (NB) new bone.

this impression to be erroneous; the sigmoid sinus, jugular bulb and petrosal veins were free of disease. The route of extension to the cavernous sinus was found to be by way of the carotid plexus of veins. Here there was evidence of a thrombophlebitis produced by extension of a suppurative process in the apex of the petrous pyramid with necrosis of the carotid canal. From the carotid plexus of veins the infection spread to the cavernous sinus by retrograde extension. Naturally the bacteremia in this case, likewise, cannot be attributed to involvement of the sigmoid sinus or jugular bulb, but must be attributed to the lesion in the carotid plexus

of veins or in the cavernous sinus. In one other case of our series studied histologically, thrombophlebitis of the carotid plexus of veins was found to be the sole cause of bacteremia. Since signs of cavernous thrombosis in the case herein reported appeared five days prior to the onset of meningeal signs, it is reasonable to assume that the meningitis was secondary to cavernous phlebitis rather than the cause of it. It is fully appreciated, however, that at times in the early stages of meningitis there may be a paucity of physical signs. Of further interest is the observation that after the chemosis, edema and proptosis of the eyes had advanced for a few days, there followed a period in the terminal stages of the illness in which these eye signs regressed. This is probably best explained on the basis that the ophthalmic vein which empties into the cavernous sinus was only partially occluded and its return flow temporarily impeded. With the re-establishment of the venous return the signs improved. Of significance from the surgical standpoint is the finding of newly formed bone in the carotid canal, which is intimately adherent to and has partially replaced the wall of the carotid artery. It can very readily be observed that in the approach to the apex of the petrosa, entailing displacement of the carotid artery as advocated by Ramadier and others, considerable difficulty would be encountered. This observation was also recently made by Guild. Finally, the bacillus Friedlander, the invasive agent in this case, is worthy of mention. This encapsulated bacillus, although by far a less frequent inhabitant of the middle ear, is, like pneumococcus type III, an organism of high virulence. I have come across no reports in the literature in which this organism was found to be responsible for thrombosis of the cavernous sinus or of the other cranial sinuses secondary to middle ear suppuration.

SUMMARY AND CONCLUSIONS.

A case of thrombophlebitis of the cavernous sinus of otitic origin, presenting interesting clinical and pathologic features is described.

While the diagnosis was readily established clinically, the route of extension to the cavernous sinus was disclosed only after histologic examination of the temporal bone in serial section had been made. The thrombophlebitis of the cavernous sinus was found to be secondary to a suppurative process

in the apex of the petrous pyramid, which had necrosed the carotid canal and had produced a thrombophlebitis of the carotid plexus of veins, the infection spreading from there by retrograde extension to the cavernous sinus.

The literature on this subject is briefly reviewed, particular emphasis being placed on the frequency of its occurrence and the routes of extension from the middle ear to the cavernous sinus.

The diagnosis of thrombophlebitis of cavernous sinus will undoubtedly be more frequently made, and the routes of extension from the middle ear to this structure will naturally be more often ascertained if careful postmortem examination with histologic sectioning of the temporal bone be routinely performed.

BIBLIOGRAPHY.

1. JANSEN, A.: Über Hirnsinusthrombose nach Mittelohreiterung. *Arch. f. Ohrenheilk.*, 35:55, 1893.
2. HESSLER, HUGO: Die Otogene Pyämie. Gustave Fischer, Jena, p. 242, 1896.
3. UTA, K.: Die Isolierte thrombose des Sinus Cavernosus bei Ohrenkrankungen. *Virch. Arch.*, 249:131, 192.
4. DWIGHT and GERMAIN: Thrombosis of the Cavernous Sinus with Report of Four Cases. *Boston Med. and Surg. Jour.*, 146:456, 1902.
5. EAGLETON, W. P.: The Carotid Venous Plexus as a Pathway of Infection in Thrombophlebitis of the Cavernous Sinus. *Arch. Surg.*, 15:275, 1927.
6. BRUNNER, H.: Beiträge zur Kenntnis der otogenen Cavernosusphlebitis. *Monatsschr. f. Ohrenheilk.*, 60:2, 1926.
7. KOERNER, OTTO: Das Fortschreiten von Krankheiten der Paukenhöhle durch den karotischen Kanal in die Schädelhöhle. *Ztschr. f. Ohrenheilk.*, 23:230, 1892.
8. SCHLANDER, E.: Der karotische Kanal als Überleitungsweg. *Monatsschr. f. Ohrenheilk.*, 63:652, 1929.
9. RICHTER, HELMUTH: Beiträge zur pathologischen Anatomie des Ohres. *Arch. f. Ohrenheilk.*, 141:334, 1936.
10. TOBECK, ALFRED: Über die Einteilung der sog Pyramiden-spitzeiterungen. *Monatsschr. f. Ohrenheilk.*, 63:654, 1929.

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ANGIOENDOTHELIOMA OF THE MASTOID. A CASE REPORT.*†

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Endothelioma, as clearly described and classified by James Ewing,¹ is applied to an extensive group of tumors which take their origin from the endothelial lining of the blood vessels, lymph vessels, and spaces such as the serous and subdural regions. Histologically, endothelium takes its origin from the mesodermal layer; pathologically, these endothelial cells may take on dual tendencies.

In the presence of inflammation they multiply, form giant cells and concentric endothelial pearls, and in granulation tissue they become ameboid and phagocytic. The endotheliomata are readily classified into four distinct groups: 1. the perivascular type, in which the endothelial cells proliferate around the blood vessel; 2. the adenoid type, in which the cells take on a cuboidal appearance and attempt to line spaces; 3. the angioendothelioma, in which the cells are both small and large, and attempt a pattern of imperfect capillaries, sinuses or even grow diffusely; 4. in which the diffuse endothelioma, where the cells are elongated, cuboidal or spindle in shape, and show a tendency to space formation. The third group, the angioendothelioma, is the type which is found in bone pathology. These grow insidiously, often without symptoms, and are first recognized by a facial paralysis and severe pain in the region of the mastoid. Later, the abducens, trigeminal, vagus, glossopharyngeal and hypoglossal nerves may be involved and extension to the brain may occur, causing a hemiplegia. A foul bloody discharge from the external canal may announce the disease.² Meningitis, brain abscess and sinus thrombosis are not complications of angioendothelioma. Roentgenologically, these tumors show irregular absorption and tend to push out the thickened periosteum, in contradistinction to osteomyelitis, which is far less destructive. Primary malignancy of the mastoid is rare. Grasser³

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reported three cases in adults. It was found only twice among 40,000 admissions to the otolaryngological department in the University of Michigan.⁴ The youngest case in the literature was one reported by Hegener,⁵ in a boy age, 3 years. Hang⁶ reported three cases, and Miodowski⁷ reported seven cases, all in adults.

Case Report: E. C. was born at the Jewish Memorial Hospital on May 11, 1938. The delivery was normal and careful discharge notes stated that he was perfectly developed. No mention was made of a facial palsy at this date. During a subsequent admission to the hospital January 28, 1939 (for a circumcision operation) the house surgeon found a



Fig. 1. Face at rest.

left facial palsy. Upon further questioning, the boy's mother stated that the condition first appeared when the baby was 4 months old. There were no complaints referable to the ears, nor was there any history of any. The child was circumcised and made an uneventful recovery. The following month, Feb. 24, 1939, the patient was again admitted to the hospital. This time the history chart read: "Infant, 9 months old, acutely ill, temperature 101°. There is slight swelling of the cervical glands. The tonsils are enlarged and cryptic. No purulent nasal discharge. The right ear shows no mastoid edema, there is a serosanguineous discharge in the canal. There is an inferior perforation of the drum. On the left side, edema is present over the mastoid, extending anteriorly to the root of the zygoma and into the temporal region. No fluctuation elicited. The canal is dry. Some edema of the superior wall of the canal present. The drum is intact, dull and bulging." On Feb. 25, 1939, the edematous

area behind the left ear was incised and pus was obtained. The local condition showed progressive improvement until March 4, 1939, when a blood count increased from 14,300 to 23,050. X-ray studies of the mastoids did not show any destruction at this time. In spite of the negative findings, the mastoid was entered, March 18, 1939. Granulations and necrotic cells were removed. Patient was discharged from the hospital on April 4, 1939; both ears and the left postauricular wound entirely healed. At the out-patient clinic, April 21, 1939, the records show that there was a serosanguineous discharge in the left external canal. Some pain was also present at this time but this symptom soon subsided. Because of the left facial palsy, stereoptican X-rays were taken for possible fracture of the skull. These proved negative. Temperature never ranged higher than 101° during this episode. Approximately five months later, we saw the patient for the first time. The boy was now 21 months old. The complaints were "something sticking out of the left



Fig. 2. Showing paresis, left side of face.

ear," as stated by the mother. Examination revealed a polyp presenting at the external opening of the left external auditory canal. It was soft and freely movable. It filled the entire external canal. Nothing else could be seen. The patient was readmitted to the hospital. Under general anesthesia, one large and one small polyp were removed by snare. Inspection, still under general anesthesia, showed that the entire posterior canal wall was absent. A large probe could be passed and moved about in the mastoid process. The drum appeared normal in color and was intact. The polyps came from the mastoid cavity and apparently had burrowed their way into the external canal through the posterior canal wall. No further surgical intervention was considered until the pathological report could be obtained. A tentative operative diagnosis of infectious granuloma was made. Specimen was sent for examination and reported by Dr. Angrist as "highly anaplastic malignant tumor tis-

sue of the angiosarcomatous type." Several pathologists who examined the slides were of the opinion that this was definitely a malignant tumor; however, they differed somewhat as to the type. We felt that an opinion should be obtained from other pathologists.

Dr. James Ewing made a diagnosis of angioendothelioma of bone. Dr. F. W. Stewart, of the Memorial Hospital, diagnosed the growth as a definite "Ewing sarcoma."

Dr. H. L. Karsner gave the following report: "In my opinion, this is definitely a malignant tumor. The only question is as to its exact nature. The infiltrative growth, the many mitotic figures, some of which are abnormal, all indicate malignancy. I can see no justification for the diagnosis of neuroblastoma, although that certainly cannot be excluded. The same applies to endothelial myeloma of bone. In one slide the arrangement and character of the cells suggests strongly a nevus,

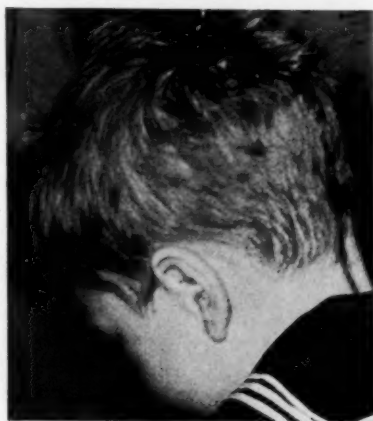


Fig. 3. Showing mastoid scar.

although no pigment is present. The other slide is made up of cells of the same general character, but the appearance is definitely more malignant, and I think this represents the malignant transformation of a nevus. The condition might properly be called, then, nevocarcinoma."

Our pathological report signed by Dr. Angrist, released March 5, 1939, stated: "Section shows overlying squamous epithelium with an area of ulceration and adjacent necrosis with compactly placed cellular tumor tissue beneath. The tumor tissue shows closely placed small, rather vesicular, cellular elements without distinct cytoplasm limitation, and minute and absent nucleoli. Numerous mitotic figures are present. Considerable variation in size and shape of cells is noted. Vascularization is present. There is hypertrophy of the lining endothelial tissue. The diagnosis is anaplastic malignant tumor tissue, other possibilities would include lymphosarcoma and neuroblastoma."

X-ray studies of the mastoid did not aid us in determining the therapeutic course to follow, nor did it show any further pathology. Because of the age of the patient, the nature of the disease and the extensive surgery already done, it was felt that radiotherapy offered the best for this child.

Beginning March 19, 1940, for just one month, the patient received 1,200 r. units of deep X-ray therapy over the region of the left ear. Oto-

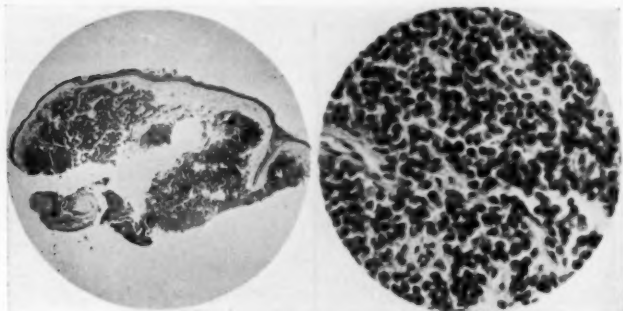


Fig. 4. High and low magnification of tumor tissue.

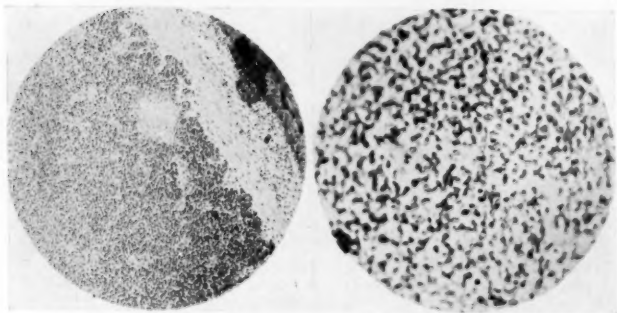


Fig. 5. High and low magnification of tumor tissue.

logical examination made September and November, 1940, showed no recurrence of the tumor, a normal drum and a large defect in the posterior canal wall of the left ear. X-rays of the long bones were negative. A sternal puncture was negative. A small gland was removed from the left side of the neck and sent to the pathologist, who reported "simple hyperplastic" lymphoid tissue with large active follicles. No invasion of malignant tissue was seen.

SUMMARY AND CONCLUSIONS.

Endothelioma of the mastoid is extremely rare. The case here presented is the youngest ever reported. The prominent symptoms were: 1. facial palsy at age 4 months; 2. a sero-sanguineous discharge; and 3. pain. Associated with this condition may be found the presence of a clinical mastoid with unusual local pathology. The only efficacious treatment is complete surgical exirpation, followed by intensive prolonged radiotherapy. The outlook, in spite of all this, is grave.

BIBLIOGRAPHY.

1. EWING, JAMES: Neoplastic Disease, 4th Ed., W. B. Saunders Co., 1940.
2. VOISLAWSKY, ANTONIE P.: Tumors of the Ear. *Ann. Otol., Rhinol. and Laryngol.*, Dec., 1905.
3. GRASSER, C.: Uber das primare Endothelium des Mittelohres bezu. Des Felsenbeins Repr. *Ztschr. f. Ohrenheilk.*
4. NEW, GORDON B.: Reprint (*Arch. Otolaryngol.*), pp. 545-552, May, 1925.
5. HEGENER: Beitrage zur Klinik unter Histologie Sarkomatoser geschwulste des Schlafenbeins Falls I Alveolarendotheliom des Felsenbeins. *Ztschr. f. Ohrenheilk.*, 37 Bd., 1900.
6. HANG: Beitrage zu Kasinstik und pathol. Histologie der Malignen Tumoren des Felsenbeins. *Arch. f. Ohrenheilk.*, 47 Bd., 1899.
7. MIODOWSKI: Zur pathologie der Schlafenbein endotheliome. *Arch. f. Ohrenheilk.*, 69 Bd., 1906.

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THE RELATION OF CHEST CONDITIONS TO SINUS
DISEASE. THE ROENTGENOLOGIST'S
POINT OF VIEW.*†

DR. KARL KORNBLUM, Philadelphia.

It is needless for one to dwell upon the importance of the subject that forms the basis of the present symposium since this fact has been repeatedly emphasized during the past decade. It is well, however, that the subject be brought to our attention at frequent intervals, that we may not lose sight of the interrelationship of the diseases affecting the various component parts of the respiratory tract. It is also important that we keep informed regarding the advances being made in the study of the relation of chest conditions to sinus disease since we are still in ignorance regarding many phases of this subject.

To have been permitted the privilege to take part in this program is the source of much gratification since it affords an opportunity to pay tribute to a Philadelphia man who, more than anyone else in this city, helped to popularize and disseminate knowledge regarding the close relationship of pulmonary and sinus pathology. I refer, of course, to Dr. Willis F. Manges, late Professor of Roentgenology of the Jefferson Medical College. This subject was one of keen interest to Dr. Manges, who was an ardent student of the respiratory tract. Much of his research and many of his writings were devoted to various diseases and conditions affecting respiration. As a testimonial of his activity in this field one may find among his writings no less than 24 papers on this and allied subjects. Dr. Manges considered the interrelationship of sinus and pulmonary disease so important that in the Roentgen examination of the chest done routinely of every student nurse in the training school of Jefferson Hospital there was included an examination of the paranasal sinuses; furthermore, in many patients in whom the history or the Roentgen examination of the chest suggested the possibility of causa-

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tive or related pathology in the sinuses, Dr. Manges would examine the accessory sinuses, although this had not been specifically requested by the referring physician. Needless to say, this was done without additional expense to the patient. The wealth of material and experience accumulated by Dr. Manges permitted him to speak as an authority on this subject. Certain it is that many of the Roentgenological practices related to the respiratory tract that are prevalent today are the direct result of his teaching, and to him the art and practice of Roentgenology is greatly indebted.

The Roentgenologist occupies an enviable position as regards the subject under discussion. Since his work brings him in daily contact with all manner of chest conditions and with most of the diseases affecting the paranasal sinuses, he must of necessity have a broad general knowledge of the anatomy, physiology, pathology and clinical manifestations of diseases affecting both the chest and the sinuses. This advantage is not shared by either the internist or the otolaryngologist. The attention of the former is likely to be centered upon the chest with the numerous affections which may involve its contents, while the otolaryngologist is concerned with diseases of the upper respiratory tract, especially the paranasal sinuses. Thus, within their restricted field of activity the relationship of cause and effect between sinus and lung pathology may possibly be missed. But the Roentgenologist is having this relationship brought to his attention almost daily, so that it becomes second nature for him to suspect the sinuses when an unexplained infection of the lungs is encountered.

When a patient complains of pulmonary symptoms, a Roentgen examination of the chest is now regarded as the most important single investigation. The clinician no longer relies upon the results of percussion and auscultation for information regarding the condition of the lungs. These have long since proven to be inadequate and unreliable when diagnostic precision is desired. It is amusing to look back upon the day when certain recognized leaders in medicine stated that pulmonary tuberculosis could not be diagnosed by the X-ray. But that day has fortunately passed and now the Roentgen examination is being used thousands of times daily to reveal the secrets of the intrathoracic cavity.

Much of the present day value of the Roentgen examination of the chest is dependent upon the marked improvement in

radiological technique that has occurred during recent years. More powerful X-ray apparatus permitting of more rapid exposures has added greatly to the definition and clearness of the various anatomical structures of the lungs. In a similar manner, improvement in intensifying screens and in the manufacture of celluloid film has added a wealth of detail to the Roentgenogram of the lungs which enable one to detect relatively small and early lesions. Thus, the report today of a negative chest carries with it a conviction that no significant pulmonary disease exists, which was not true 10 or 15 years ago.

What are the conditions in the chest that the Roentgenologist considers etiologically related to the paranasal sinuses? (See Table I.) First and foremost is the acute respiratory

TABLE I.
ASSOCIATED DISEASES OF THE PARANASAL SINUSES.

1. The Acute Respiratory Tract Infection.
The "Common Cold."
2. Chronic Tracheobronchitis.
Bronchosinusitis.
3. Chronic Pulmonary Emphysema.
4. Bronchiectasis.
5. Bronchial Asthma and Hay Fever.

tract infection, the so-called "common cold," which often manifests itself Roentgenologically as an acute tracheobronchitis. Occasionally this may result in a pneumonia, of either the lobar or bronchopneumonic type. In the patient with recurring upper respiratory tract infections, one looks for evidence of a chronic tracheobronchitis. Chronic pulmonary emphysema is often associated with a chronic sinusitis, and in bronchiectasis the etiological relationship to the sinuses is well recognized. Finally, one must give due consideration to bronchial asthma and hay fever.

If one considers each of these clinical entities from the Roentgenological point of view, the relationship of the pulmonary to the sinus disease becomes quite apparent. It has been amply shown by various investigators that the "common cold" is not a localized affair limited to the nose, pharynx, larynx, trachea, bronchi or lungs, even though one or more of these structures may give rise to the predominating symp-

toms. It is now recognized that in the "common cold" there is an inflammatory involvement of the entire respiratory tract. The infection that starts as a sore throat soon ascends to the nose and sinuses, and concomitantly descends to the larynx to give hoarseness, and then proceeds to the trachea and bronchi, resulting in a very troublesome cough for a few days. In many instances a careful physical and Roentgenological examination of the chest would reveal a patch of pneumonia which ordinarily goes unrecognized since the vast majority of individuals with a "common cold" do not have medical attention. It is only when the pulmonary complication is severe that a physician is called and the diagnosis of pneumonia is established.

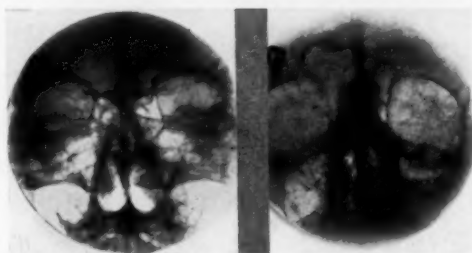


Fig. 1. Acute sinusitis limited to the left maxillary sinus. Note the fluid level indicating the presence of an exudate in the left antrum.

To appreciate the extent of the involvement in the "common cold" one need only make a Roentgen examination of the paranasal sinuses and chest. In the sinuses (see Fig. 1) one will often find an opacity of one or more of the group of cells which tends to clear as the symptoms from the "cold" subside. In the Roentgenogram of the chest (see Fig. 2) there will be found in many instances unmistakable signs of an acute tracheobronchitis, manifested by accentuation of the hilum and trunk shadows of the lungs. In the Roentgenogram one sees only the changes occurring in and around the bronchi, so that, strictly speaking, the Roentgen appearance is that of a bronchitis. The changes in the trachea are not seen Roentgenologically. Nevertheless, it has become customary to speak of the condition as a tracheobronchitis since it is generally recognized that the two conditions go hand in hand. This has been amply verified by bronchoscopic examination.

Evidence of sinusitis and tracheobronchitis may not be present at one and the same time but each can usually be demonstrated at some time during the attack. Of course, in the acute respiratory tract infection one cannot necessarily say that the pulmonary disease is the result of the sinus involvement. The entire tract by continuity becomes involved in an inflammatory process, which may originate in any part of it, usually the nose or throat. It is important, however, to recognize this universal involvement of the entire respiratory tract during the



Fig. 2. An acute tracheobronchitis manifested by an increase in the prominence of the hilum and trunk shadows of both lungs. This can frequently be demonstrated in the ordinary "common cold."

course of an ordinary "cold" so that when symptoms persist beyond the time usually allotted to such an infection one may be led to scrutinize carefully every part of this tract for the residual infection acting as a focus and preventing complete recovery. Under such circumstances one must strongly suspect the paranasal sinuses since they are ideally situated and anatomically constructed to harbor infection which is often difficult to eradicate completely.

The individual left with a chronic sinusitis from a "common cold" then becomes predisposed to the development of any one of several pulmonary conditions. Acting as a focus from

which infection may disseminate every time the individual is exposed to inclement weather or when his physical resistance is below par, he becomes the subject of frequent "colds." With each there is an acute flare-up of the sinusitis and the development of an acute tracheobronchitis. The condition in the trachea and bronchi becomes chronic and as a result of the peribronchial inflammatory reaction and fibrosis that occurs there is noted Roentgenologically a progressive intensification of the hilum and trunk shadows of the lungs. In contrast to the indistinct, blurred and only moderately opaque shadows of these structures in acute tracheobronchitis, in the

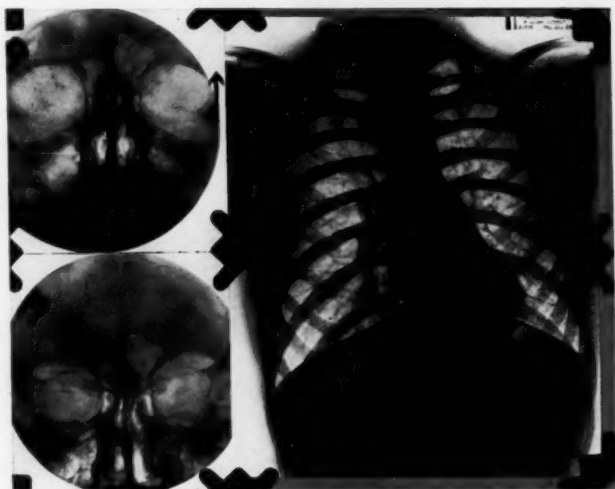


Fig. 3. Chronic bronchosinusitis showing chronic infection of the left maxillary sinus and a chronic tracheobronchitis of the lungs. Such a patient is subject to frequent attacks of acute respiratory tract infection usually emanating from the chronic disease in the sinuses.

chronic form the shadows increase in density, become sharper and more distinct and involve a greater portion of the lung field. The shadows radiate outward from the hilum into the lung parenchyma in all directions but show a tendency to be more marked in the bases. The recognition of this relationship between chronic sinusitis and chronic tracheobronchitis has given rise to the term bronchosinusitis (see Fig. 3). This designation is particularly suitable since it immediately directs attention to the dependence of the pulmonary symptoms upon chronic disease of the paranasal sinuses.

While the condition may never progress beyond the stage of a bronchosisinusitis, in many instances there develops one of two conditions, or a combination of the two; namely, chronic pulmonary emphysema or bronchiectasis. As a direct result of the chronic cough that is an inevitable part of a bronchosisinusitis, there develops a gradual and progressive expansion of the pulmonary alveoli. This alveolar dilatation causes a gradual increase in the lung volume and brings about certain changes that are well recognized as characteristic of chronic pulmonary emphysema. Among these are the enlargement of the thoracic cage, giving rise to the so-called

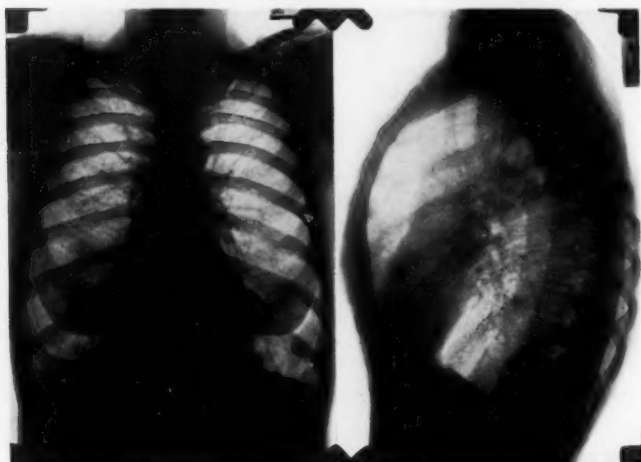


Fig. 4. Chronic pulmonary emphysema. The characteristic changes are well shown in the lateral view of the chest.

barrel-shaped chest. This is accompanied by a rounded kyphosis of the thoracic spine and a flaring of the lower ribs. In addition, there is considerable depression of the domes of the diaphragm. These changes are quite readily recognized Roentgenologically (see Fig. 4). The barrel-shaped enlargement of the chest, with increase in the width of the intercostal spaces, the overaeration of the lung fields, with diminution in the density of the hilum and trunk shadows, the depressed diaphragm, which in extreme cases seems almost to be inverted, the restricted diaphragmatic movement

as noted fluoroscopically, the rounded kyphosis and increase in the preaortic space as seen in the lateral view of the chest produces a picture that has become a classic in Roentgenology. From the clinical side, in addition to the chronic cough, which gradually becomes nonproductive, there develops a progressive dyspnea which comes to be the predominate and, in many instances, a most disabling symptom.

One of the most tragic sequelae of a chronic bronchosisinusitis is the development of bronchiectasis. The frequent recurrence of pulmonary infection resulting from a bronchosisinusitis brings about an extensive peribronchial fibrosis. This tends to distort the bronchi and diminish the elasticity of the pulmonary tissues. Thus, the patient experiences increasing difficulty in eliminating the normal and abnormal pulmonary secretions. Stasis invites further infection. The chronic inflammatory process thus resulting produces a weakening of the bronchial walls, which then become dilated and eventually terminate in the clubbing and sacculation so characteristic of a well established bronchiectasis. Gravity plays an important part in the localization of the disease predominately in the bases of both lungs. As in the case of many of our most malignant diseases, bronchiectasis develops slowly and insidiously. In most instances, before the condition is even thought of, the patient has developed the well known symptoms of a chronic cough with expectoration of large amounts of fetid, purulent material, especially on arising in the morning. This denotes an advanced case of bronchiectasis.

From the Roentgenological point of view the diagnosis of bronchiectasis often presents a difficult problem. In the advanced case the appearance is easy of recognition (see Fig. 5). Engrafted upon the usual Roentgenological manifestations of a chronic tracheobronchitis there is to be noted a predominance of heavy trunk shadows at both bases. These shadows are no longer sharp and clean-cut but have become hazy and ill-defined. Small patches of increased density may stud the basilar portions of the lungs. When these become so numerous and so large as to merge, there results a more or less homogeneous density, through which the heavy trunk shadows may be but dimly visible. These changes in the bases are the result of retained secretions in the dilated and sacculated bronchi, although they may be accentuated by an accompanying inflammatory involvement of the surrounding lung

and adjacent pleura. This picture is readily diagnosed by all Roentgenologists as bronchiectasis and needs only to be confirmed by the bronchoscopist.

It is in the early and developmental stages of bronchiectasis that Roentgenology often fails in the recognition of the condition since in this stage there may be little or no Roentgenological evidence; however, the Roentgenologist who has this condition constantly in mind will often suggest the prob-

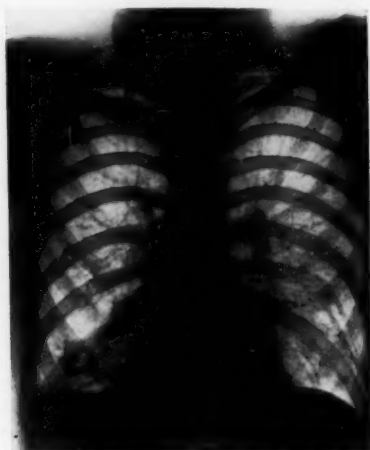


Fig. 5. An advanced case of bronchiectasis. This is readily recognized in the Roentgenogram.

able existence of a bronchiectasis, even though the Roentgenogram may show but little evidence of the disease. A correlation of the clinical history with the Roentgenological manifestations of a severe chronic tracheobronchitis should lead one to suspect a bronchiectasis. This is especially true if there has been established the coexistence of chronic disease of the paranasal sinuses since it is generally recognized that chronic sinusitis is the most frequent predisposing cause of bronchiectasis.

When bronchiectasis is suspected and the Roentgenogram fails to give conclusive proof of its presence, pneumonography is indicated; that is, the injection of an opaque oil into the bronchi for the purpose of visualization of the bronchial tree.

This will usually establish the diagnosis even in the earliest cases by showing slight but definite dilatation of the bronchi (see Fig. 6). In the more advanced cases, pneumonography is not required for diagnosis but is often used to determine the extent and localization of the morbid process.

The recognition of bronchiectasis is of vital importance to the patient. I have come to regard bronchiectasis as a far more serious disease than pulmonary tuberculosis. With our modern methods of prevention, detection and treatment of



Fig. 6. Pneumonography showing a slight dilatation of the lower lobe bronchi, especially on the left side. This is an early case of bronchiectasis of the type that is not readily recognized in the plane film of the chest.

tuberculosis, this disease has been brought fairly well under control. Bronchiectasis, on the other hand, is insidious in its onset, progresses so slowly without producing any very alarming symptoms, and is often not recognized either clinically or Roentgenologically that it frequently has progressed to an advanced stage before the diagnosis is finally established. While the majority of cases of tuberculosis heal spontaneously, bronchiectasis probably never does so. The symptoms of bronchiectasis are often mistaken for tuberculosis. When repeated sputum examinations fail to show tubercle bacilli and Roentgenograms of the chest are negative for tuberculosis, the condition is likely to be passed off as just another case of

bronchitis, no thought being given to the possibility of a bronchiectasis. Not until the patient is troubled with a fetid, purulent expectoration is the condition suspected and the patient sent for Roentgenological and bronchoscopic examinations. The disease has then reached an advanced stage and while modern surgical methods may eventually salvage these unfortunate individuals, it must be admitted that our greatest hope in bronchiectasis at present is prevention rather than cure. The situation has been well summarized by Boyd,¹ who says: "When bronchiectasis is well established in childhood an early death from bronchopneumonia can only be regarded as a blessing." Thus, it becomes imperative that we recognize the fact that chronic disease of the paranasal sinuses is the most common factor that initiates and keeps active a bronchiectasis. To treat bronchiectasis without attempting to eradicate or at least to lessen the infection in the sinuses is futile. In this connection it needs to be emphasized that a clinical investigation of the sinuses is not sufficient, since it is generally recognized that the Roentgen ray often detects significant disease in the paranasal sinuses that is either not suspected or is missed on clinical investigation. It must be admitted that the reverse is also true. It becomes important, therefore, that suspected cases of bronchiectasis be given a thorough and careful examination by both the otolaryngologist and the Roentgenologist.

It is well to direct attention to the fact that the variety of bronchiectasis resulting from a chronic sinusitis is almost invariably bilateral and is limited or at least predominates in the base of the lungs. When a bronchiectasis is found to be unilateral and localized to one lobe, especially an upper lobe, the etiology is usually not to be found in the paranasal sinuses (see Fig. 7). Under such circumstances the condition is more apt to be the result of a foreign body or the sequel of an unresolved pneumonia or a lung abscess. This is an extremely important differentiation since the prognosis is much more favorable. Treatment consists of removal of a foreign body, if present, and the establishment of drainage by dilating a stenosed bronchus, and doing repeated bronchoscopic aspirations. In the more advanced cases, lobectomy can be performed. Since the disease is a purely localized affair and unilateral, the morbid process can often be completely eradicated. This is in sharp contrast to those cases dependent

upon a chronic sinus infection, since here the entire respiratory tract shows evidence of a chronic inflammatory process with varying degrees of tracheobronchitis, pulmonary emphysema and bronchiectasis. The disease, therefore, cannot be entirely eliminated.

As regards these localized forms of bronchiectasis, it becomes necessary to call attention to the fact that when the disease is of some duration, but more particularly when it is quite severe, a Roentgen examination of the paranasal sinuses will often reveal evidence of a chronic sinusitis. One

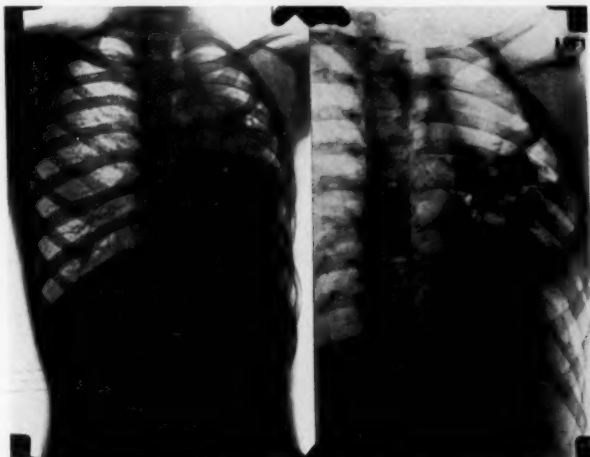


Fig. 7. A unilateral and well localized advanced bronchiectasis involving the left lower lobe. This type of bronchiectasis does not usually result from chronic sinus disease.

should not, however, jump to the conclusion that the pulmonary disease is secondary to the sinus infection. As a matter of fact, the reverse is more likely to be the case. With a chronic suppurative lesion in the lung, the constant cough and expectoration keeps the upper respiratory tract bathed with purulent material. This brings about a chronic inflammatory process and leads to frequent reinfections of the sinuses. We have become accustomed to think of these chronic respiratory tract infections as always emanating from above, with the sinuses usually the initiating culprit. It is well to keep in mind this retrograde method of dissemination of

infection from the lung, to bronchi, to trachea, to pharynx, to nose and then, finally, the sinuses. This reversal of the process is also important in the usual bilateral basal bronchiectasis of sinus origin since in these cases infection also tends to spread from the bronchiectatic lung upward to the sinuses, thus creating a vicious circle.

Finally, it becomes necessary to consider sinus disease in relation to bronchial asthma and hay fever. An individual who is allergic may show evidence of this phenomenon in the

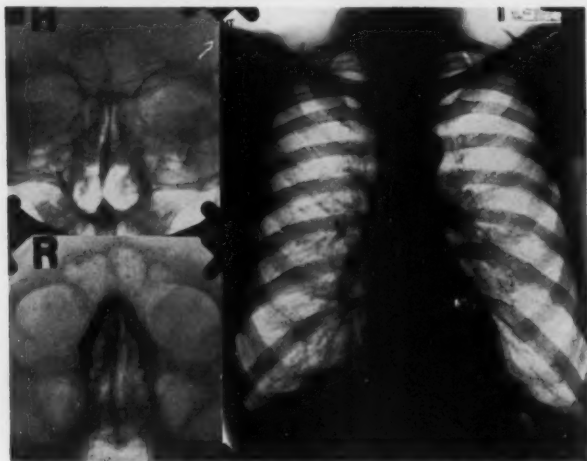


Fig. 8. This patient has had bronchial asthma for a number of years. The Roentgen studies show chronic infection of the ethmoid and maxillary sinuses with chronic tracheobronchitis and emphysema of the lungs.

nose, the lungs or simultaneously in both. The vasomotor changes that occur in the nose and sinuses, and the bronchial spasm occurring in the lungs predispose these structures to infection. It becomes common, therefore, to find victims of asthma and hay fever who also exhibit chronic infection of the nose and sinuses and a chronic tracheobronchitis of the lungs. Such pathology is usually quite readily demonstrated by Roentgen examination (see Fig. 8), although in the case of the sinuses the problem presented may be a difficult one. The Roentgen findings do not always permit of a differentia-

tion between a chronic infection and the changes resulting from allergy. This differentiation is best made by the otolaryngologist. It is possible, however, for the Roentgenologist to suspect allergy from a Roentgen examination of the sinuses. In patients who give a history of chronic sinus disease for a number of years and in whom Roentgenograms show not the slightest evidence of pathology, one can suspect that allergy may be at the bottom of the difficulty. Clinical investigation will often show that the individual is definitely a victim of allergy.

The Roentgenologist can often suspect bronchial asthma from the appearance seen in Roentgenograms of the chest. Since the pathology of bronchial asthma consists of a thickening of the walls of the smaller bronchi, with small areas of emphysema, the appearance produced on the Roentgenogram is precisely the same as that of a chronic tracheobronchitis. Confronted with such a picture, one cannot make the differentiation between the changes due to infection from those due to asthma. Of course, in many instances there is a combination of the two conditions present. In considering possible etiological factors for the Roentgen findings of a chronic tracheobronchitis, asthma must always be suspected, and this possibility should be reported to the referring physician.

During an attack of bronchial asthma, a Roentgenogram of the chest may present a startling picture. Changes simulating a severe bronchopneumonia may be found. These changes are transitory and rapidly disappear with subsidence of the attack. In the usual case of asthma the changes already described for chronic recurring infections of the respiratory tract will be found. According to Manges and Hawley,² approximately 63 per cent of cases of bronchial asthma show definite Roentgenological changes in the lungs.

Mention should be made of the fact that a chronic sinusitis may in itself initiate a state of allergy, while the sinus infection occurring as a complication in asthma and hay fever may tend to aggravate the existing allergy; furthermore, a chronic infection of the respiratory tract, dependent upon a primary allergic state, may develop along the lines already outlined for chronic bronchosinusitis and its manifestations may come to dominate the clinical picture, its importance often superseding that of the initiating hay fever or bronchial asthma.

SUMMARY.

In this presentation there is briefly discussed the relationship of sinus disease to chronic infections of the lungs. Originating in the "common cold," perpetuated as a chronic sinusitis, there is established a predisposition to recurring infections of the respiratory tract, with a resultant chronic bronchosinusitis. This may eventuate into chronic pulmonary emphysema or bronchiectasis. The relationship of chronic infections of the sinuses and lungs to the allergic states of bronchial asthma and hay fever is considered. Special attention is given to the importance of Roentgenology in the detection and localization of infections of the sinuses and lungs. The Roentgen examination offers our best means of demonstrating the dependence of these infections one upon the other.

REFERENCES.

1. BOYD, WILLIAM: The Pathology of Internal Diseases. Lea and Febiger, Philadelphia. 1935. 2nd Ed., p. 181.
2. MANGES, W. F., and HAWLEY, S. J.: Roentgen Findings in Chests of Asthma Patients. *South. Med. Jour.*, 20:2:126-134, Feb., 1927.
3. MANGES, W. F., and HAWLEY, S. J.: Roentgen Ray Observations in Asthma. *Jour. A. M. A.*, 89:870-872, Sept. 10, 1927.

1020 Sansom Street.

HEARING AND HEARING-AIDS — A REVIEW OF THE RECENT LITERATURE.

DR. GORDON BERRY, Worcester, Mass.

I. — INTRODUCTION.

Hearing Aids continue to occupy the interest, and an increasing one, of otologists, hard-of-hearing (hypacusic) patients and acoustic engineers. It is to the last group that we owe the remarkable progress of the past decade. First, it was the medium of the telephone that offered the amplification employed, and the hypacusic patient used the steadily improving Carbon Aid. Then the Radio came into general use, and the Vacuum Tube Aid had the advantage of the research being applied to this industry. With the advent of war, telephonic, radio and electrical workers and supplies will be in demand for military use. After this emergency is over, the research efforts on acoustics exerted in the military field will be applied again to the civil field and the science of making and using hearing aids will advance correspondingly.

Recent literature on hearing aids is of four divergent types, corresponding to the four groups concerned with their manufacture and use: the hypacusic individual, the otologist, the acoustic engineer and the psychologist. Each group uses its own language, which in some instances is difficult for other groups to understand.

II. — THE INCIDENCE OF DEAFNESS.

1. *The World's Fair Test*: Two articles give interesting data as to the prevalence of deafness. The first is entitled "The World's Fair Hearing Tests." This was the first extensive public test for musical tones. It was conducted by the Bell System at both New York and San Francisco. The tones used were 440, 880, 1,760, 3,520 and 7,040 cycles; 550,000 records were obtained.

Interesting findings were: There is an increasing impairment with advancing years, a high tone loss showing more in

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the men, and a low tone loss showing more in the women. A hearing loss of 25 dcb. at frequencies up to 1,760 cycles begins to be a handicap. The individual will usually be aware of such an impairment, and will experience difficulty in understanding speech under conditions of public speaking, such as in the church or theatre or around the conference or dinner table. The distribution curves show that only about 1.5 per cent of the young people taking the test, or three out of 200, have a hearing loss of 25 dcb. or more for tones of these frequencies. In the oldest age range (50-59 years) almost 10 times as many, or every seventh person, show this much impairment.

A hearing loss of 45 dcb. for frequencies up to 1,760 cycles will usually make it difficult to hear direct conversation even when the speaker is two or three feet away. Individuals with this much loss usually need some sort of hearing aid.

Acuity for the two high frequencies (3,520 and 7,040) is less important than for the low tones (440, 880), so that a loss for the high tones is not such a serious handicap. High tone deafness is of particular interest to the extent that it is indicative of a progressive condition which may later involve tones of lower frequency. Of the people taking the World's Fair tests, some 6 per cent in the 20-29 year age range showed a hearing loss in excess of 25 dcb. for the 7,040 cycle tone. In the 50-59 year group, half of the people showed such a loss. It is thought the average of the country's population would run worse than this, because the World's Fair groups represented an average or better economic state, and the lower economic groups show a greater prevalence in hearing defects.

The tests were elaborately conducted and cross-checked, and the elements of error were reduced to an insignificant minimum.

2. *United States Health Service Test:* Beasley² reports on the nationwide survey conducted by trained workers of the United States Health Service in 1935 and 1936. Among other health items, entries were made on the degree of deafness. Five classifications were used: 1. Partial deafness, stage 1: difficulty in understanding speech at a distance or in group conversation — later determined to be 46 to 48 dcb. loss for

1,024 and 2,048 cycles, respectively. 2. Partial deafness, stage 2: difficulty with direct conversation at close range, can hear over the telephone: 55 dcb. loss. 3. Partial deafness, stage 3: difficulty in using telephone, but can hear amplified speech: 68 dcb. loss. 4. Total deafness for speech. Cannot hear speech, but acquired his own capacity to speak before losing his hearing: 89 dcb. loss. 5. Deafmute. Born deaf or lost hearing before speech was acquired. — Those who reported good hearing averaged a 7 dcb. loss.

The survey was done by sampling average localities. A total of 2,502,391 persons were interviewed, or 3.7 per cent of the whole United States population in 1930.

One out of every 78 males and one out of every 85 females had a loss of 47 dcb. (first stage). Children (age 5 to 14 years) showed one out of every 889 males, and of every 442 females, with 47 dcb. loss. At 35 to 40 years, these figures were one out of 103 males or 108 females; in the 65 to 74 year period, one out of every 14 males or 18 females.

The age at which deafness comes on is shown by these figures. Those per 100,000 population showing first stage impairment, 45 dcb. loss are 25 in the 5 to 14 year group, six in the 15 to 24 year group. After this drop, there is an annual acceleration, with a peak at 35 to 44 years, then a retardation, followed by another period of acceleration beyond the age of 55 years.

Lastly, a consistently higher prevalence of impaired hearing occurs in the lower income groups (below \$1,000 per year per family as against over \$3,000 per year). The low income persons show 25 to 112 per cent more incidence of deafness than obtains in the higher income group. Of those under 25 years of age in the low income group, one of every 273 males or 349 females showed stage 1 deafness or worse; of the higher income group, one out of every 404 males or 613 females had a similar impairment.

III. — EMPLOYMENT OF THE HARD-OF-HEARING.

1. The possibilities of the modern hearing aid have their repercussions on employability. Dr. E. P. Fowler³ points out that while previously an 80 dcb. loss in hearing indicated a total loss of usable hearing, and a person with such loss would

be considered unemployable from the hearing standpoint, now, because of better hearing aids, this no longer holds true. The important frequencies for speech are 256 to 4,096 cycles. A properly fitted hearing aid can give adequate hearing to those with an 80 db. loss in some of these tones. If such adequate correction can be secured, the act of wearing a hearing aid should place no greater limitation on a man's employability than the wearing of glasses.

2. As evidence that the wearing of a hearing aid offers no employment handicap, an editorial in the *Volta Review* quotes Vincent P. Maher,⁴ Deputy Superintendent of Los Angeles Schools, as assuring all teachers and other employees of the school board that the board heartily approves the use of a hearing aid whenever there is any need. There is no thought of discrimination against those who wear glasses or false teeth, and there is absolutely no thought of discrimination against the use of hearing aids. The only ones who need feel censure are those who do not avail themselves of the wonderful new devices offered to those who have impaired hearing.

3. This same editorial quotes a report by Leon Brady, Ph.D., Research Associate in the Centre for Safety Education in New York University, on the Safe Operation of Motor Vehicles. As far as hearing is concerned, there did not appear to be any convincing evidence that persons who are partly or entirely deaf cannot drive safely.

4. Under the title, Does Your Job Depend on Hearing?, Dr. William E. Grove reports on 43 replies to a questionnaire sent to employers of labor in all types of work and occupations. He concludes: With the exception of a few special trades, such as telephone operators, stenographers, proof readers and salesmen, there is no kind of work which the person who is hard-of-hearing cannot do and do well if he approximates his psychological attitude to the normal, if he compensates for his defect with lip-reading and hearing aids, if he makes himself just a little more efficient than his fellow, and if he is sober, industrious and honest. Industry will not employ him because of his defect but in spite of it.

5. In a similar paper, at the 1941 Central Zone Conference of the American Society for the Hard-of-Hearing, Grove⁷ adds: In regard to employer's ratings as to the occupational

success of the deaf and the hard-of-hearing, the report of the United States Department of the Interior Bulletin No. 13 is very interesting. These ratings were under four different headings:

Succeeding very well	54.1%
Succeeding fairly well	32.0%
Getting by	11.2%
Failing	2.7%

Those with a higher education or with special trade training fared better than those with no such training.

IV. - LEGISLATION AND CONTROL.

1. *Council on Physical Therapy*: Howard A. Carter,* Secretary of the American Medical Association Council on Physical Therapy reported concerning the Council's investigation of hearing aids and audiometers. This investigation began six years ago when vacuum tube aids were not yet available. The hearing aid industry is about 40 years old. Although vast strides have been made, the perfect aid has not appeared on the market. At the present time, only in a general way, which is far from being precise, can a hearing aid be fitted to an individual. It is something like buying a ready-made overcoat: there are several standard sizes in the store and one size of overcoat will fit a large number of individuals without much alteration. When electrical hearing aids have been in existence 400 years, like spectacles, it may be possible to fit them with the same precision.

This Council was formed to examine and pass on audiometers and hearing aids. Its personnel includes most of the leaders in this field: Dr. Bunch, Dr. Coates, Dr. Fowler, Dr. Grove, Dr. Jones, Dr. Macfarlan, Dr. Nash, Dr. Newhart, Dr. Shurly and Dr. Wherry, together with Dr. Paul Sabine, of the Riverbank Laboratories, and Dr. W. C. Beasley, of the U. S. Public Health Service.

Certain requirements for hearing aids are set up; the manufacturers submit to the Council and their expert consultants the instruments and the data concerning their performance. This material is passed on by impartial experts and the decision arrived at as to whether the instrument meets the requirements. There are now (June, 1941) 17 aids on the Council's list of accepted devices: seven carbon, 10 vacuum, two semiportable.

The advisory consultants are of the opinion that hearing aids should be acquired only after a complete otological examination. The physician is in a better position to analyze the wants of a deafened patient than a technician or salesman, who in most instances lives on the commission from the sales. The otologist will know whether lip-reading instruction, a surgical operation or the application of a hearing aid is the correct way to solve the patient's problem, and he is the best able to evaluate its efficiency and performance for the patient.

2. *Licensing of Audiometrists:* Crutchett⁹ discusses the fitting of hearing aids in successive numbers of the *Volta Review*. First, he presents possible regulations governing those practicing audiometry, in order to raise to a proper level the standard of those now so engaged. Then he offers suggestions for a course of not less than two years which all practicing audiometry must take. Some of the subjects proposed are physics, communication engineering, vacuum tube amplification, audiometric testing, psychology, mechanism of hearing, training of residual hearing, lip-reading, mechanism of speech, phonetics, historical and current literature on audiometers and hearing aids.

3. *Comments on Licensing:* The *Volta Review*¹⁰ offers several comments by representative men concerning Mr. Crutchett's proposals on the training and licensing of audiometrists. A representative of one of the hearing aid manufacturers favors it. A representative of another company says: "The major companies spend many thousand dollars yearly in an effort to train their personnel—not merely so that their sales may be increased, but so that hearing aids may be sold on a better basis, so that customer satisfaction is greater—to the ultimate end that the general acceptance of hearing aids shall be greater. We think that very little of practical value could come out of the conclusion that these people were not well qualified for the work they are doing. We believe, therefore, that the proposition of combining the work of the audiometrist and the work of the individual who fits or sells the hearing aids can profitably remain open, at least until further data have been accumulated."

Dr. Newhart commends the interest shown but feels that the dealer is likely to be too interested in making a sale, and, therefore, the initial audiometric review and the

final approval might better be in the hands of the impartial otologist.

Dr. Douglas Macfarlan welcomes this activity and thinks that to be reliable and effective it should work under the aegis of some national otological society. Others speak for and against. Some dealers feel that the personal psychological phase enters so much into the fitting of aids that the exactness of audiometry, as here proposed, will not prove adequate.

Halstead¹¹ claims that the fitting of deafened and hard-of-hearing patients can be done only by specialized persons. Acoumetrists should be trained and licensed just as optometrists are.

4. *The Fitting of a Hearing Aid:* In another article, Crutchett¹² considers the case of a figurative Mr. Jones, who goes to a hearing aid dealer for a fitting. If possible, Mr. Jones should already have gone to an otologist, secured all the therapeutic help that otology could offer. He should secure from his otologist the names of acceptable instruments and reliable dealers in his community, and take to a dealer the otologist's recommendation as to an instrument. This recommendation should indicate which ear is to be fitted, and whether an air or a bone conduction terminal is to be used. It should be in writing and should include any other findings which may be of help to the dealer, such as a discharging ear that should not be plugged with an ear mold, or growths or other conditions of the canal that would make an ear mold hard to fit. From the audiogram chart and from data supplied by the manufacturer, the dealer should then select the aid, combination of parts, or adjustment that is best suited to the needs of Mr. Jones. This fitting should then be checked by an audiometric test of the hearing while the aid is worn, and by articulation tests. Finally the user should try the instrument for a week, at home, at business and at church. If he is satisfied and if the otologist approves, Mr. Jones should buy the hearing aid. If he is not satisfied, he should seek the help of some other dealer.

The author adds that both otologists and dealers often fail to have the proper equipment or the proper knowledge fully to participate in this fitting program. Only some of the otologists have audiometers and only a few of them know

enough about hearing aids and the methods, personality and ability of the dealers to enable them to make worth while recommendations; also, only some of the hearing aid dealers have audiometers and only a few know enough about them to know how to use them. Not all manufacturers supply their dealers with data as to the output of their aids so that any attempt can be made to select the instrument indicated by the audiogram. Facilities are available for this type of accurate testing. The hard-of-hearing public should expect otologists and dealers to be supplied with the knowledge and equipment to do their part of the fitting correctly and completely.

V. - AUDIOMETRIC TESTING.

1. *Essential Frequencies:* Audiometric measurements for correlated hearing losses were reported by Beasley¹³ on 16,620 ears. He concludes:

a. Correlation between hearing losses by air conduction is extremely high and regression is linear for the four tones, 64, 128, 256 and 512 cycles. For both screen and clinical types of audiometers, any one of these four tones will provide approximately equal predictive value as to acuity of hearing throughout this range. For greater simplicity of audiometer design, a single pure tone near 500 cycles is preferable.

b. Correlation of hearing losses on 1,024 and 2,048 cycles is higher for females than for males. Since either tone is about equally predictive of the ability to hear speech, a single tone in the region of 2,000 cycles is preferable.

c. Correlation is lower, dispersion is greater and regression more irregularly curvilinear for hearing losses on pairs of tones higher than 1,024 cycles than for lower tones. Consequently, in hearing tests of high tone loss, more than two tones should be provided at levels above 2,048 cycles.

Comment: Then Beasley's audiometer for screening or clinical use would have four frequencies; 500 cycles, 2,000 cycles and at least two at higher levels.

2. *Range of Bone Conduction Hearing:* In an article on the Limits of Audition by Bone Conduction, Watson¹⁴ finds the total audible frequency range for open canals by bone conduction to be 25 to 17,000 cycles, which is much greater

than was earlier supposed possible. The maximum usable intensity for open canals was tentatively determined at 80-90 db. and at 1,000-2,000 cycles.

The same author¹⁵ presents the results of careful experiments showing that the hearing of speech by bone conduction may attain as high a degree of perfection as that attained by the more usual method of air conduction. The maximum performance was gained when the user's mouth was closed, his teeth touching but not clinched, and his ear canals occluded.

VI. - ACOUSTIC RESEARCH.

The acoustic engineer publishes an admirable and scholarly monthly entitled, the *Journal of the Acoustical Society of America*, dealing with details of acoustics and electrophonics, with noise phenomena and with musical instrument performances, with transmission systems, sound reflections and speech peculiarities, to name a few of the subjects discussed in its pages. Harvey Fletcher¹⁶ gives an article on Loudness, Masking and the Process of Hearing which is pertinent to the subject we are reviewing. It requires a peculiar technical knowledge to understand the graphs and charts and mathematical formulae appearing in the ensuing 18 pages, in which he shows how to define loudness and loudness level in a quantitative way. In particular, the data from the masking effects of thermal noise were used to calculate the relation between the position of maximum stimulation on the basilar membrane and the frequency of the tone producing the stimulation.

This reference is pertinent to the subject under discussion and is given to point out to the otological reader what care and accuracy is shown in the performing and reporting of the elaborate research work done by the acoustic engineers; to demonstrate how their background and viewpoint is different from ours; and to suggest that the otologist without such a background or training will have to rely on the acoustic engineer for a complete understanding of the mechanics of the hearing aid and its proper application to a hearing deficiency.

VII. - CARBON VS. VACUUM TUBE AIDS.

Knudsen¹⁷ gives the results of careful comparisons between the carbon and vacuum tube type of hearing aid. He says his

results were similar to Ewing and Ewing, who tested four hard-of-hearing individuals with both types of hearing aids. With the speaker 12 inches away, the carbon aid gave a 38 per cent response to the average syllable articulation test, while the vacuum tube aid gave a 72 per cent response. With the speaker 40 inches away, these responses were 17 per cent and 67 per cent, respectively, not only showing greater efficiency in the vacuum tube aid but at a greater distance as well.

Still more benefit accrues from a binaural fitting. An illustrative case, with the speech articulation test at 18 dcb. above his threshold, gave a 60 per cent response with one aided ear, and a 70 per cent response with the other aided ear. Then earpieces were applied to both ears (with a single microphone and a single amplifier), and the binaural response was 85 per cent. The binaural ideal is a separate fitting: two microphones, one for each ear.

As compared with monaural hearing, binaural hearing (with one microphone) possesses these advantages: 1. an increased loudness level of about three phons (dcb.), which in certain cases of nerve impairment is highly desirable; 2. a better resolving power, both in respect of frequency and intensity; 3. greatly improved sense of sound location; 4. less sensitivity to the disturbing effects of room reverberation; 5. greater ability to focus attention on the source of sound and to disregard extraneous noises.

Comment: It will be obvious why two separate instruments, one fitted for each ear, would offer advantages; but why a single microphone connected to both ears helps so much may not be as clear. The sense of fitness and perspective probably comes from the use of both the right and the left brain centres, giving a familiar balanced acoustic response instead of a unilateral one. Only the very hard-of-hearing are willing to go to the extra trouble of wearing earpieces in both ears.

VIII. — EAR DEFENDERS.

Closely associated with hearing aids is the use of some type of earplug to protect the acoustic nerve against damaging noises. Knudsen¹⁸ has given this matter much thought. It is

more important than has been generally granted, for our industry is increasingly noisy, and the noise from gun fire and shell explosions must now be considered. One study revealed that 75 per cent of the workers in one boiler factory were unable to hear at all at a public meeting, or could only hear with difficulty.

Tests with a noise frequency of 512 cycles gave the following sound-insulating properties for different kinds of ear defenders: cotton stuffed in the ear, 7 dbc.; cotton rolled in a tight taper, 12 dbc.; wax or wax and cotton mixtures, 25 to 35 dbc.; the various rubber stoppers, 25 to 35 dbc. It must be borne in mind that these stoppers offer no insulation to bone conducted sounds through direct contact with vibrating floors or hand drills. The wearing of these stoppers helps conversation in this noisy environment, for it reduces the masking element of the prevailing noise. But all those conversing must wear these ear defenders, for those wearing them will not talk as loudly, and then those not wearing them will be unable to hear.

In an earlier article, Knudsen¹⁹ gives further details, emphasizing the cumulative shock to the nervous system from noise. He feels that stoppers, to offer more than a 25 to 35 dbc. impedance, should be designed on acoustic damping principles. Different acoustical elements of impedance can be assembled in series. A simple application of this principle is a double-walled partition in which the two septums are loosely coupled by means of flexible separators or air spaces. Knudsen has devised one upon this principle. It is essentially a tapered rubber tube containing an outer plug of heavy metal and an inner plug of soft rubber. They are coupled by an air space between, and the enclosing rubber walls of the tube. These must be so inserted that both the outer and the inner plugs close off the canal. This defender by audiometric test provides an insulation factor of 50 dbc. against air-borne noise.

Comment: These ear defenders are being made in quantity.

IX. — HEARING AIDS AS HELPS.

1. *In Lip-Reading:* There is an old idea that lip-reading ability will cause the lip-reader to use the hearing less, and so let it diminish from lack of use. Miss Rankin,²⁰ Executive

Secretary of the Des Moines Society for the Hard-of-Hearing, claims this is an exploded fallacy. The student of lip-reading should find his use of residual hearing greatly increasing as his lip-reading ability reaches the point of easy, relaxed lip-reading. Nonused residual hearing does tend to slip away, but the use of lip-reading brings the deafened individual back into conversation, where he makes an effort to both see and hear.

Miss Rankin favors the use of hearing aids but feels that the combined use of hearing aids and lip-reading yields yet better results. By learning to read lips, with hearing as well, sound memory is preserved and speech is kept normal.

2. *In Speech:* Mrs. George B. Katzenberger,²¹ of Indianapolis, carries this idea into speech training. In working with individuals who are hard-of-hearing, it helps greatly in correct speech production to have them use a properly fitting hearing aid. Such words as cap — cat, leaf — leave, cave — gave, look the same to the lip-reader and can be distinguished through this hearing aid.

A REVIEW OF THREE BOOKS.

Comment: In order to give the reader better access to the current literature on hearing aids, three recent books have been reviewed. It is hoped that the material and purpose of the authors has been fairly presented so that the interested and busy otologist can adequately inform himself from this digest, while he who seeks detailed knowledge will thus know where to go for the particular material he desires. To obtain a more complete review, some repetition has been employed.

X. — ADVICE ABOUT HEARING AIDS.

In a 156-page book entitled, *A Handbook of Hearing Aids*,²² Niemoeller offers primary information to the user and to the otologist concerning these instruments and their use. He finds that any hard-of-hearing person who can understand speech shouted slowly, directly into his ear, has an excellent chance of being fitted satisfactorily with a hearing aid. He thinks that more difficult than the selection and proper maintenance of the instrument is adapting oneself to the new and confused sounds that now reach the ear. With the improve-

ment in these aids, he urges the hard-of-hearing not to forget the importance of the other two important helps: otological care and lip-reading.

The essential parts of a hearing aid are described: transmitter (the microphone), receiver (the ear terminal), battery, cord and amplifier. The usual transmitter uses carbon granules. A crystal transmitter employs electrodynamic chemical crystals (many use Rochell crystals), which are low in acoustic output but when used with vacuum tube amplifiers can be made very effective, thus correcting many of the faults of the older carbon sets. He feels that for low grades of hearing impairment the carbon aid, with its peak effectiveness placed where most needed, makes the best small, light, compact aid, requiring low amplification and small battery consumption. But the vacuum tube amplification is truer, has less buzzing or other noises, and gives greater amplification. It is a heavier and more expensive unit.

The dealer is described as discovering the best aid for his customer by employing an assembled instrument that permits differing combinations of varying parts of hearing aids, through the use of control switches. The combination giving the best hearing is finally selected for the customer. The customer, on the other hand, should seek an instrument to meet his present needs, not a future more profound deafness. He should secure sufficient amplification, but not carry more weight and pay more money for amplification he does not need. It is urged that several different instruments be tried before the final selection is made, and that the advice of the otologist and dealer be given great weight, for the patient has used his hearing so little that he does not know just what selective amplification he had best secure. He must be cautious against high-pressure salesmanship, and place little reliance on performance in the ideal surroundings of the dealer's office. A trial period before purchase should be insisted upon. A moderate charge should be made for this trial, to be credited against the price if the instrument is purchased.

In the chapter on the use of the hearing aid, the author urges its employment when deafness has reached a 20 per cent loss level, and says that lip-reading should also be employed as a supplement to the aid. Instructions in the care and maintenance of hearing aids are given.

The last chapter deals with the regrettable sensitivity of the average hard-of-hearing individual against wearing an instrument. In catering to this sensitivity, one manufacturer hides the cord in a string of ornamental beads, and another builds the hearing aid inside of a set of false teeth.

XI. — THE TESTING AND FITTING.

One of the most helpful recent articles on hearing aids is a 30-page monograph entitled, *Audiometers, Hearing Aids and the Ear Specialist*, by Mr. L. A. Watson,²³ who generously permits this review. Realizing how important audiometric graphs are for the proper fitting of hearing aids, part of the manuscript dwells on audiometry.

A. — *Audiometers.*

1. *Standardization of Audiometers:* Audiometers are calibrated against a standard maintained at the Bureau of Standards. All instruments must reach this level of efficiency to gain the approval of the Bureau or of the American Medical Association Council on Physical Therapy.

2. *Soundproof Room:* Variations in the technique of using the audiometer will cause more variation in the resulting readings than will the difference between a reasonably quiet room in a doctor's office and a soundproof room. It is to be remembered that the low frequency tones are masked much more by surrounding noises than are high tones (from 1,000 cycles up); also, that a patient with say a 30 db. loss of hearing is not affected by a prevailing noise of 10 to 20 db.

3. *Injury:* The most likely injury to an audiometer comes from dropping or damaging the receiver. It is urged that the otologist calibrate his own audiometer by making check audiograms of the same three or more normals at least once a year.

4. *Pointers in Audiometric Testing:* Most audiometrists have the patient face away from the audiometer; shutting the eyes also increases concentration. The receiver must be precisely over the canal. When testing high tones it is well to shift the receiver occasionally, because high tones have a tendency to create a standing wave that may cause a change of intensity as much as 10 db. The threshold rating should

be taken at the point where an increasing tone is first heard. This reading will be 5 to 10 dbc. more exact than that taken at a point where a diminishing tone is first lost.

5. *Maximum Intensities:* There is a maximum intensity which is variable for different tones. At 128 cycles, the greatest loss one can measure is 70 dbc. Any higher amplification would cause pain. This is the maximum required by the Bureau of Standards. At 256 cycles, the maximum is 80 dbc. For 500 to 4,000 cycles, the maximum is up to 100 dbc.

6. *Loudness Balance:* There is a difference in the apparent loudness of a given tone when heard by a perception deafness case as against the apparent loudness of the same tone for a conduction deafness case. This has an important bearing on the ability of the patient to endure amplified sounds through his hearing aid. In the conduction deafness case there is uniformity of loss for all levels at or above his threshold. For instance, a person with a 40 dbc. loss just begins to hear at this threshold. A tone of 50 dbc. intensity sounds the same to him as a 10 dbc. tone does to the normal ear; a 70 dbc. intensity sounds like a 30-40 dbc. tone to the normal; and a 100 dbc. sound is not deafening but is the same as a 40-60 dbc. noise to the normal. The normal can simulate the sound heard by this hard-of-hearing person by partly or completely shutting off his ear.

Contrast this with a perception (nerve) deafness case with a 40 dbc. loss. Amplify the test tone from 40 up to 50 dbc. and the sensation is not that of a 10 dbc. sound but more as a 30 or even 40 dbc. sound to the normal ear. And a 90 dbc. sound seems as loud to this patient as it does to the normal. This is the so-called recruitment factor. The use of this loudness balance offers one way to differentiate between the conduction and the perception type of deafness.

7. In this connection, Mr. Watson agrees with Mr. Snyder, of the Bureau of Standards, that no pure conduction deafness case has a loss of over 50 dbc. Beyond this point there must be some perception (nerve) deafness superimposed. This can be demonstrated by plugging tight the ear canals of a normally hearing individual, thus creating artificially a pure advanced conduction deafness. Now test him with the audiometer by air conduction. At about 40 dbc. the increas-

ing tone will again be audible. This argument is used to show that bone conduction aids should rarely, if ever, be recommended for any loss of more than 50 or 55 db.

B. — Speech Sound Frequencies to Be Amplified.

In trying to discover the proper hearing aid correction for a given type of deafness, it is helpful to know the different ranges used for speech. Mr. Watson places the man's voice at 128, while a woman's range is an octave higher, or at about 256 cycles (middle C). But the tone or pitch of the voice is not the most important element in speech. The sound components between 500 and 3,000 cycles are the most essential for speech intelligibility. This is why perception deafness cases hear the voice readily enough but cannot understand the words spoken. This critical range of speech intelligibility may be broken down as follows: vowels and fundamentals, 100 to 800 cycles; liquids and nasals (l, r, m, n), 500 to 2,000 cycles; dentals and spirants (s, t, p, th, etc.), 1,800 to 3,000 and higher.

One understands from this why a nerve or senile deafness case with the high tones curtailed hears the spoken voice at from 100 to 300 cycles readily enough but cannot distinguish the speech essentials over 2,000 cycles (such as p, b, s, sh and d). The most essential tones for the entire speech range are at 1,000 and 2,000 cycles. In this range, an estimated 17 per cent of all persons over 65 years of age have a hearing loss of at least 40 per cent.

In the range from 500 to 3,000 cycles, a loss of 20 db. gives but little trouble. At 40 db. a handicap is experienced and a hearing aid should be considered. A 40 to 85 db. loss can be corrected or greatly benefited by a well fitted hearing aid. A 100 db. loss can be marked off as beyond the help of an aid.

A very helpful chart is given here, through Mr. Watson's courtesy.

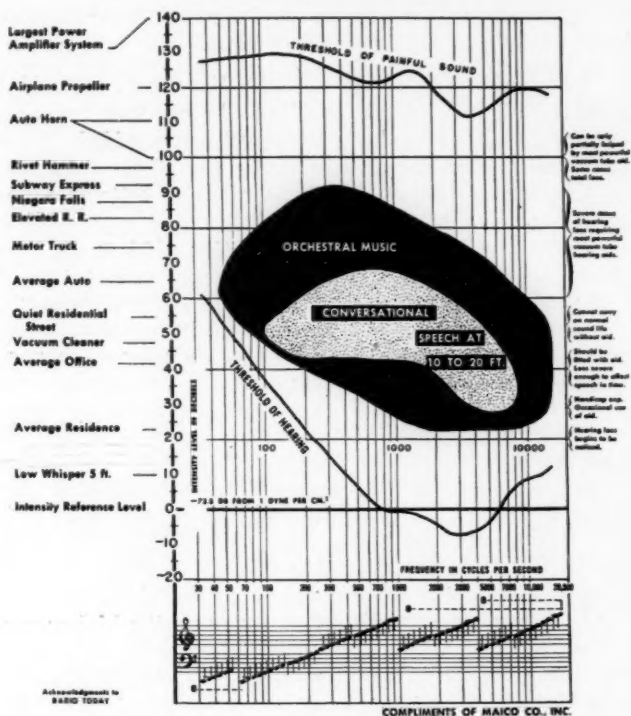
C. — Hearing Aids.

1. *Preference in Hearing Aids:* A hard-of-hearing person who has fair hearing in certain tones, especially the lower tones, and poorer hearing in others, especially the higher tones, at first prefers a hearing aid that accentuates the

sounds he hears best. The high pitched elements have been lacking for him, and he has come to think of speech as not containing these sounds. When proper amplification gives him back these high sounds, he objects to it as being too bril-

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liant, too loud, unnatural. In short, he desires an amplification which actually is distorted but which seems true to him. This case needs a re-education of his hearing. It may be best not to give a full correction for high tones at first. When he is adjusted to this change, then a higher correction will be more welcome, working toward the ideal of a fully corrected

capacity throughout the pitch range. Performance will be better the more nearly this latter goal is finally attained. Here, the otologist can explain and guide. The carbon aid gives its greatest peak of amplification at around 1,000 to 2,000 cycles, and then drops off sharply. It is in this middle range that the average nonrehabilitated individual first desires the amplification. But with training, he learns that his hearing is more efficient if selective amplification is applied. This can best be secured, especially in the high tones, by the more powerful and wider ranging vacuum tube aid.

2. *Which Ear Should Be Fitted?* It may be difficult to determine which ear should be fitted. An ear that has a loss of less than 50 db. is giving some direct help unaided. Therefore, fit the poorer ear whenever possible, if the better ear has a loss of less than 50 db. This will give better binaural balance, sound will be easier to localize and there will be better depth and focus to sound perception. If both ears are much impaired, it is best to advise an instrument in each ear. A binaural fitting increases the hearing gain from 7 to 10 db. over that enjoyed by a monaural fitting and gives a bilateral sense of hearing. But most people prefer not to be encumbered with the extra earpiece.

3. *An Unmasked Fitting — Binaural:* The British have been using binaural fitting to secure what is called "unmasked hearing." It helps advanced perception deafness cases. The objective is to give the full range of tone in one ear, but to the other only pure high tones are delivered. The reason for this is that with some perception and nerve types of deafness the predominance of low and vowel sounds becomes so strong that it tends to mask out or cover up the higher consonant sounds which they most need. Having the full range on one ear gives a feeling of naturalness, while the giving of pure highs to the other ear provides an intelligibility for the consonant sounds, and does make a surprising difference in many of these peculiar, severe and advanced cases of nerve loss.

4. *Air vs. Bone Conduction:* Another question is whether air conduction or bone conduction will serve better. Formerly, when only carbon aids were available, there was no suppressive action enjoyed. They had a sharp peak of extra

loudness around 1,000 cycles. The user, with the amplification increased to hear the soft low or high tones of speech, would experience discomfort or pain when a 1,000 cycle amplified sound of the same or greater intensity was heard. These users found that a bone conduction receiver absorbed this over-amplification at the 1,000 level and were willing to sacrifice the greater distance and perception of the air conduction receiver in order to gain the freedom from painful loudness afforded by the bone conduction receiver. Usually a relative increase in the bone conduction test is found to be present more in the low tones and less present in the high tones. A bone conduction receiver will amplify the low tones more; however, low tones tend to mask out high tones, while high tones do not mask out the low tones. The net result is a masking out of the high tones in the range where hearing is already diminished. Only if the audiometric test shows the bone conduction to be at least 20 or 30 db. better than the air conduction will a bone conduction receiver prove more efficient.

5. *Power Output:* Amplification has been discussed. This is the first consideration in a hearing aid. Another important characteristic is what is called "power output." This is the striking force behind the amplification and is measured in milliwatts of acoustic power. The perception cases of deafness need more power output and less amplification than the conduction deafness cases. An illustration may be found in the radio field. A small radio turned up high will give much noise but the sounds will seem tinny and distorted, too loud, and even indistinguishable; while a large and powerful radio can be turned to a low amplification but the sounds will be clear and distinct, even at some distance. This power output is an important characteristic of a good hearing aid.

6. *Electric Current:* Battery economy is another important characteristic. This was an early fault of the vacuum tube aid which has been largely corrected through refinements in the midget tubes.

7. *Selective Suppression:* Selectivity is a characteristic secured in recent models. This is done by different types of filters. By these filters, the low or the middle or the high tones may be prevented from coming through. In this way the desired selective amplification is secured. The hearing

aid amplifies all pitches. To amplify enough where the hearing is weak will increase far too much the tone frequencies where the hearing is good. The introduction of the proper filter will reduce the too loud tones, favor the weaker tones and give the level amplification desired.

Today selective suppression can be incorporated in the vacuum tube aid. This permits the fainter sounds to be amplified, while it prevents the excess of loud noises to come through. The clapping of hands and similar loud noises near the instrument is no longer painful because of this dampening effect on overloading sounds. The dampening gained by a bone conduction receiver is no longer needed, and the greater efficiency and better performance of the air conduction receiver makes it usually the preferred terminal. This suppression factor also helps in those cases of perception deafness where there is a strong recruitment factor: for example, where an 80 db. sound is just as loud to them as it is to the normal ear, without amplification, and where a 40 or 50 db. sound is not loud enough to be heard and needs amplification.

8. *Error in Personal Preferences:* In fitting a case with a hearing aid, particularly a case with good low tone and weak high tone perception, it will be found that the individual user will not accept a full correction of his high tones. He is not used to hearing them, they sound unnatural, he finds them loud and confusing. This case should first be given a partial correction. When he is used to this, he will welcome a stronger emphasis of the high tones.

9. *Speech Test:* A speech perception test that distinguishes the low frequency vowels from the middle frequency liquids and nasals (l, r, w, m, n), and these from the high frequency dentals and spirants (t, th, d, s, z) will demonstrate where the hearing acuity is lacking in speech element perception, and also whether the hearing aid corrects that lack.

10. *The Earpiece:* Yet another essential characteristic of a good air conduction hearing aid is a well fitting earpiece. If this insert does not fit snugly, there is a sound leakage, with a resulting whistle or "microphonic feed back." Such an earpiece also provides good bone conduction through the contact with the lateral walls of the ear canal. The insert

should go in as far as the cartilaginous canal does. Otologists are urged to familiarize themselves with the technique of making a plaster mold of the ear for their patients.

The lucite earpiece is very sturdy and will not break, nor is it affected by heat or cold; however, alcohol will affect it and should not be used to clean the earpiece. A pipe cleaner serves well for keeping clean the hole through the insert.

D. — Resulting Performance.

1. *Amplification Gained:* Mr. Watson claims that with the new selective amplification it will be possible to step up any band of frequencies, either low or high, as much as 50 to 60 db. This is made possible by a new small vacuum tube unit which is so powerful that two tubes will amplify as much as three of the earlier tubes did. Also, these tubes are sturdier and can stand more injury. Another way to show this gain is to contrast the modern tubes with the English tubes first used in 1933 and 1934. Three of these in a unit drew 210 mills, while the present tubes draw 54 mills for a similar unit. Thus the latter produces more power with much less battery consumption.

2. *Speech Is Helped:* Speech is maintained at its best level by a well fitting aid. Where hearing for certain elements of speech is lacking, the speech tends to become slurred in those same elements, because the individual can no longer check his own performance. This is especially true for a lack in the high consonant range. A hearing aid that restores the hearing in this range corrects the speech fault.

XII. —THE PSYCHOLOGY AND PHYSIOLOGY OF AUDITION.

In 1938 was published an important book on Hearing by Stevens and Davis,²⁴ of Boston. In the preface these authors say that within a decade the field of audition has undergone impressive transformation, and even while the manuscript was being written, the subject had expanded beyond the confines of the single volume proposed, so that the relation of the science of audition to architecture and applied acoustics, to speech and phonetics, to the problem of noise, and to music were some of the deliberate omissions. This negative view gives an idea of the thoroughness with which the recent advances in the science of acoustics and audition are pre-

sented in this 450-page volume. The first half deals with the psychological aspects, providing the student with the fundamentals of the science of sound. It answers the question, "What do we hear when we listen?" The second half deals with the physiology of hearing, telling the student "how he hears when he listens." The book impresses the reviewer as the best assembly of recent scientific work on audition that he has seen. It has been carefully reviewed and abstracted for the reader's benefit. But a lack of space necessitates its deletion.

BIBLIOGRAPHY.

1. STEINBERG, MONTGOMERY and GARDNER: *Jour. Acous. Soc. of Am.*, p. 291, Oct., 1940.
2. BEASLEY, WILLIS C.: *Jour. Acous. Soc. Am.*, p. 114, July, 1940.
3. FOWLER, EDMUND P.: *Hearing News*, p. 4, Sept., 1941.
4. MAHER, VINCENT P.: *Volta Rev.*, p. 736, Dec., 1941.
5. BRADY, LEON: From an editorial in *Hygeia*, Nov., 1941.
6. GROVE, WILLIAM E.: *Hygeia*, p. 895, Nov., 1941.
7. GROVE, WILLIAM E.: *Proc., Central Zone Conference, Am. Soc. Hard of Hearing*, p. 34, June, 1941.
8. CARTER, HOWARD A.: *Proc., Central Zone Conference, Am. Soc. Hard of Hearing*, p. 69, June, 1941.
9. CRUTCHETT, RALPH: *Volta Rev.*, June-July, 1941.
10. NEWHART, H., and MACFARLAN, D.: *Volta Rev.*, Comments by Aurex Corp. and Radio Ear, Aug., 1941.
11. HALSTEAD, THOMAS H.: *N. Y. Jour. Med.*, p. 352, Feb. 15, 1941.
12. CRUTCHETT, RALPH: *Volta Rev.*, p. 546, Sept., 1941.
13. BEASLEY, WILLIS C.: *Jour. Acous. Soc. Am.*, p. 104, July, 1940.
14. WATSON, N. A.: *Jour. Acous. Soc. Am.*, p. 294, April, 1938.
15. WATSON, N. A.: *Jour. Acous. Soc. Am.*, p. 90, Oct., 1937.
16. FLETCHER, HARVEY: *Jour. Acous. Soc. Am.*, p. 275, April, 1938.
17. KNUDSEN VERN O.: *Jour. Acous. Soc. Am.*, p. 29, July, 1939.
18. KNUDSEN, VERN O.: *Hearing News*, p. 2, Sept., 1941.
19. KNUDSEN, VERN O.: *Jour. Acous. Soc. Am.*, p. 32, July, 1939.
20. RANKIN, ELIZABETH: *Proc., Central Zone Conference, Am. Soc. Hard of Hearing*, p. 106, June, 1941.
21. KATZENBERGER, MRS. GEORGE B.: *Proc., Central Zone Conference, Am. Soc. Hard of Hearing*, p. 110, June, 1941.
22. NIEMOELLER, A. F.: *Handbook of Hearing Aids*, Harvest House, New York.
23. WATSON, L. A.: *Audiometers, Hearing Aids and the Ear Specialist*. A monograph furnished through the courtesy of the Maico Co., Minneapolis.
24. STEVENS and DAVIS: *Hearing*, John Wiley and Sons, New York, 1938.

36 Pleasant Street.

IN MEMORIAM

ROBERT LIVINGSTON LOUGHRAN, M.D.,

1873-1942.

Dr. Robert L. Loughran, well known otolaryngologist practicing in New York from 1903 until his retirement in 1934, died at his home in Sharon, Conn., Jan. 28, 1942, at the age of 68 years.

Graduating from Princeton University in 1895, he received his medical degree at the College of Physicians and Surgeons, Columbia University, in 1899. From 1903 until 1914 he was associated in otological practice with Dr. James F. McKernon, of New York. He was formerly assistant surgeon of Manhattan Eye, Ear and Throat Hospital; attending otologist to and faculty member of the Post-Graduate Medical School and Hospital; assistant otologist to the Lying-In Hospital. In the World War, Dr. Loughran held the rank of Major in the Medical Corps of the U. S. A., serving in the Panama Canal Zone, where he was also Chief Public Health Officer of the Panama Canal.

He was a Fellow of the American Laryngological Association, the New York Otological Society, and from 1924 to 1936 served as secretary of the American Laryngological, Rhinological and Otological Society, where his outstanding service and his sympathetic and charming personality endeared him to the members of that Society.

After his retirement from practice, in 1934, he occupied himself largely with carpentry, weaving and other hobbies.

He is survived by his widow, Althea H. Briggs Loughran, to whom we extend our sincere sympathy. L. G.

BOOK REVIEW.

Applied Physiology of the Nose. By Arthur W. Proetz, A.B., M.D., Professor of Clinical Otolaryngology, Washington University School of Medicine. Cloth, octavo, XXII+, 395 pages and 91 illustrations, St. Louis, Annals Publishing Co., 7200 Wydown boulevard, 1941. Price \$7.00.

Some years ago the *Jour. A. M. A.* published a small book on the art and practice of medical writing. It is a valuable little book. It seems to be unknown to many writers of articles and books in the field of rhinology. For these as a rule are badly written, full of meaningless statements and numerous misstatements and are apt to bear evidence of improper reasoning from inadequately ascertained facts. And it is the generous fashion of the reviewers of such articles and texts to comment only in compliment. Whatever the motive, the result too often is to obscure the truth.

It is this reviewer's good fortune to be asked to review a book on rhinology which is singularly free from these common defects. The composition is clear. The useless sentence is hard to find. Redundancy and ambiguity have been avoided. Facts are plainly stated and the reasoning is logical.

This book by Dr. Arthur W. Proetz is to be recommended as being the first publication of merit on the subject of the applied physiology of the nose. The author presents his work to the profession in all humility, for he has written as a rhinologist for rhinologists, with the ardent hope that his sympathetic understanding of the requirements in this field may partially compensate for his limitations as a physiologist.

This book contains much that will interest the pure physiologist. For the great mass is new in the sense that at present it is not to be found in certain standard works on physiology. For instance, one of the largest and most used textbooks on physiology covers the physiology of the nose in one-half page of fine print. Many references are given to works on the physiology of the nose by physiologists as well as by rhinologists. The large literature covered has been wisely dealt with, for the great mass of facts and conclusions of these articles has been quite rightly ignored by the author. The physiologist who reads this book will do well to ponder this fact. Dr. Proetz discussed these articles with many rhinologists of long experience and from the medley of opinions has had the wisdom and common sense to clear his way to sane conclusions. In this period of wild abandon in writing on medical subjects he has appreciated that it is the quality of an article and not the amount of ink spilled that matters.

This book can be recommended to the practicing rhinologist as being full of facts of the greatest practical value. There are 18 chapters on the following subjects: the functions of the nose, — historical notes, — structure as a basis for function, — the sense of smell, — the rigidity of the nasal structure, — air currents in the nose, — air pressures, respiration and air exchange, — humidification, — healing, — electrical surface charges, — four chapters on the cilia, — other defenses of the mucosa, — vascular and lymphatic reactions, — neural reactions, — climate, environment and other extrinsic influences.

Following these are two chapters on the clinical applications of nasal treatment and of nasal surgery which are practical considerations based upon the facts of the preceding chapters.

Many theories of function to be found in the larger rhinological textbooks are critically reviewed and some of these seriously questioned. In consequence, doubt is thrown upon the efficacy of several popular forms of treatment. It is very evident that Dr. Proetz attaches great importance to the preservation of the activity of the cilia of the nose. No one reading this book

can fail to realize that the nose is a physiological organ of the highest importance to the individual and, consequently, when dealing with a pathological condition in the nose the surgeon must endeavor to have the final result interfere as little as possible with normal physiological activity.

Such considerations, however, must not be permitted to lead to the wholesale condemnation of all current surgical procedures in the nose. For the surgeon of experience has seen such gross pathological conditions in the maxillary sinus that he will utterly refuse to subscribe to the notion that a few drops of ephedrine in the middle meatus will result in cure.

The large bibliography which follows each section will be found useful but must hardly be considered complete or well chosen. References have been included for which the author—to judge from his own text—has but scant respect. The inclusion of so many names may have been wise for political reasons but such considerations have no place in a scientific textbook. The quality of the writing or work should be the only criterion. There is a regrettable lack of reference to certain carefully written articles from the British Isles.

The book is worthily dedicated to Johnathan Wright (1860-1928), a great New York pathologist and laryngologist, whose wisdom and early advice regarding the surgery of the nose have too long been neglected.

Dr. Proetz's book already has had the effect of arresting that avalanche of nasal surgery which has done so much to discredit a great specialty. The book is a great and worthy contribution to sound rhinolaryngology and merits the very careful consideration of every rhinologist.

D. E. S. W.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The following officers in the Los Angeles Society of Ophthalmology and Otolaryngology were appointed for 1942:

President: Dr. John Osburn.

Vice-President: Dr. Gil J. Roberts.

Secretary-Treasurer: Dr. Colby Hall.

Committeeman: Dr. Harold Mulligan.

Place: Los Angeles County Medical Association.

Building: 1925 Wilshire boulevard, Los Angeles.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

DIRECTORY OF NATIONAL OTOLARYNGOLOGIC SOCIETIES.

American Otological Society.

President: Dr. E. M. Seydell, 107 W. Douglas Avenue, Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73rd Street, New York.
Place: Atlantic City, May 28-29, 1942.

American Academy of Ophthalmology and Otolaryngology.

President: Dr. Ralph I. Lloyd, 14 8th Avenue, Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce Street, Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Building, Omaha.

American Broncho-Esophagological Association.

President: Dr. W. Likely Simpson, 899 Madison Avenue, Memphis.
Secretary: Dr. Paul Holinger, 1150 N. State Street, Chicago.
Place: Atlantic City, June 8-9, 1942.

American Laryngological Association.

President: Dr. T. E. Carmody, 227 16th Street, Denver.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th Street, New York.
Place: Atlantic City, May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbitt, 1912 Spruce Street, Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Avenue, New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Place: Atlantic City, June 1-3, 1942.

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology.

Chairman: Dr. Gordon F. Harkness, 215 Main Street, Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust Street, Philadelphia.

Pacific Coast Oto-Ophthalmological Society.

President: Dr. Ralph A. Fenton.
Secretary: Dr. C. Allen Dickey.
Meeting: May 11 to 14, 1942; Portland, Ore.

Los Angeles Society of Ophthalmology and Otolaryngology.

President: Dr. John Osburn.
Secretary-Treasurer: Dr. Colby Hall.
Place: 1925 Wilshire Boulevard, Los Angeles, 6:00 P.M., Fourth Monday,
Monthly, September to May, Inclusive.

